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Renal Artery Stenosis in Patients with Peripheral Artery Disease: Prevalence, Risk Factors and Long-term Prognosis

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WHAT THIS PAPER ADDS

The presence of renal artery stenosis (RAS) is variably reported in patients with peripheral arterial disease (PAD), mostly incidentally revealed when imaging the lower extremity arteries. However, its potential prognostic significance has been poorly addressed in this setting. The present data suggest that patients with PAD present a non-negligible rate of RAS. Nevertheless, the concomitant presence of RAS and PAD do not lead to a worse prognosis.

Objective/Background: The objective was to determine the prevalence and clinical determinants of renal artery stenosis (RAS) in patients undergoing digital subtraction angiography (DSA) for the assessment of peripheral artery disease (PAD), and to evaluate its prognostic significance.

Methods: All DSAs performed from January 2000 to January 2006 were retrospectively reviewed for assessment of PAD in patients naive for any prior revascularisation of lower-limb arteries. All DSA studies were read by two senior physicians blinded to outcome, and consensus was reached in cases of disagreement. RAS was defined as the presence of $\geq$50% stenosis in either renal artery. Patients' electronic medical files were systematically reviewed and follow-up was completed by contact with family physicians until January 2014. The primary outcome was composite, including death, peripheral revascularisation, or any limb amputation. Secondary outcomes were all-cause mortality, and another composite, including death and non-fatal myocardial infarction or stroke or coronary or carotid revascularisation.

Results: In total, 400 consecutive patients having a first DSA of lower extremities, two thirds of whom were for critical limb ischaemia, were studied. Thirteen patients were excluded owing to poor renal artery imaging. RAS was detected in 57 patients (14%). Only two factors were independently and significantly associated with RAS in multivariate analysis: diffuse PAD (involving both proximal and distal segments [odds ratio {OR} 3.50, 95% confidence interval {CI} 1.16–10.54; p = .026]) and decreased glomerular filtration rate (OR 0.55 per 30 mL/minute/1.73 m^2, 95% CI 0.41–0.75; $p < .001$). During follow-up (mean ± SD 62 ± 47 months), 25% experienced limb amputation and 54% died. In multivariate analysis, no significant association was found between RAS and primary outcome (hazard ratio 0.80; 95% CI 0.57–1.10). No significant association was found with secondary outcomes.

Conclusion: Incidental RAS is frequent (14%) among patients with PAD undergoing lower extremity imaging. No difference in outcome in patients with RAS versus those without RAS was seen. Larger studies are necessary to draw definite conclusions.

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INTRODUCTION

Patients with lower extremity peripheral artery disease (PAD) present an altered long-term prognosis, mainly because of fatal and non-fatal cardiovascular events, mostly related to widespread atherosclerosis affecting different vascular beds, as well as comorbidities such as chronic kidney disease (CKD).[1–6]

Several studies have reported increasing prevalence of renal artery stenosis (RAS) with age, mostly related to atherosclerosis in the elderly.[2] Prevalence is also increased in the presence of cardiovascular risk factors, and atherosclerotic disease in other vascular beds. There can be a long period of occult progression leading to renal failure and

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hypertension, but in many cases, it may remain silent and only be detected by imaging. The presence of RAS is variably reported in patients with PAD, mostly incidentally revealed when imaging the lower extremity arteries.[7–14] However, its potential prognostic information has been poorly addressed in this setting.[11,14]

In this study, the aim was to determine the accurate prevalence of RAS and its clinical determinants in patients with PAD hospitalised for revascularisation, and to determine its prognostic significance, regarding both limb and the patient outcomes. It was hypothesised that concomitant RAS is frequent among these patients and associated with increased risk of death and cardiovascular events.

MATERIALS AND METHODS

Patient enrolment and baseline data collection

All digital subtraction angiography (DSA) studies of lower limb arteries performed between 1 January 2000 and 31 December 2005 at Dupuytren University Hospital, Limoges, France, for assessment of atherosclerotic PAD were reviewed retrospectively. The patient list was obtained via interrogation of the university's imaging database. The study was limited to patients who were naive for prior revascularisation and who underwent their first angiogram. Also excluded were DSAs performed prior to intervention for aneurysmal or inflammatory disease. The first aim was to determine out any correlation between the distribution of PAD (i.e., proximal or distal or diffuse PAD) and outcome, and the results have already been reported elsewhere.[15] All DSA studies were visually assessed by two senior physicians (V.A. and P.L.) who were blinded to patient outcomes, and consensus was reached in case of disagreement. They determined the presence/absence of a $\geq$50% stenosis in renal arteries, as well as any artery of the lower limbs. RAS was defined as the presence of $\geq$50% stenosis in either renal artery. Renal arteries were visualised during retrograde aortography after femoral puncture, without renal catheterisation. Notably, none of these patients underwent renal revascularisation.

Medical charts were studied to collect baseline risk factors and comorbidities which were defined as: patients were considered hypertensive if they took any antihypertensive drug for this purpose and/or if their average systolic or diastolic blood pressure exceeded, respectively, 140 mmHg or 90 mmHg during the first two blood pressure measurements after admission. Diabetes was defined by a fasting blood glucose $\geq$7 mmol/L on admission or the use of any oral antidiabetic agent and/or insulin. Hyperlipidemia was defined according to the documented patient history and/or a fasting blood cholesterol $\geq$240 mg/dL. Smokers were defined as active cigarette smoking ever, at baseline, or in the past. Coronary artery disease was defined according to documented medical history and/or any history of coronary revascularisation. Heart failure was defined according to the documented medical history and/or in the presence of New York Heart Association functional class III–IV dyspnea. Cerebrovascular disease was defined by any documented episode of stroke, transient ischaemic attack, or carotid revascularisation. CKD was defined as end stage renal disease with dialysis, or a glomerular filtration rate (GFR) $<$ 60 mL/minute/1.73 m^2 calculated according to the Modification of Diet in Renal Disease formula.[16] Critical limb ischaemia (CLI) was defined according to the TransAtlantic InterSociety Consensus II criteria.[17]

Follow-up

Patients' medical computerised files were systematically reviewed until January 2014, and follow-up was completed by telephone contact with family physicians when the latest actual clinical contact was $>$1 year previously. Events noted during follow-up were death, fatal and non-fatal myocardial infarction or stroke, and coronary or carotid revascularisation. The primary outcome was composite, including death, peripheral revascularisation, or limb amputation (major or minor), whichever came first. Two secondary outcomes were defined. The first considering all-cause mortality, and the second a composite, including death and non-fatal cardiovascular events (myocardial infarction, stroke, or coronary or carotid revascularisation).

Statistical methods

Data are reported as mean $\pm$ SD and *n* (%) for continuous and categorical variables, respectively. These variables were compared in patients with versus those without RAS, using the Student *t* test and Fisher's exact test, respectively. A logistic regression model, using the stepwise downward method, was run to determine variables independently associated with the presence of RAS. Kaplan–Meier survival method was used for the comparison of survival according to the presence of RAS, using the log-rank test. The follow-up index was calculated as recommended.[18] Multivariate analysis was performed to assess whether RAS was independently associated with outcomes, using a Cox proportional hazards model. Variables with a univariate *p*-value $<$ .2 were selected for further multivariate analysis. Special care was taken to avoid colinearity and only variables with the best univariate *p*-value were incorporated into the multivariate analyses of those with particularly close clinical meaning (e.g., insulin treatment and diabetes). In all analyses, $p < .05$ was considered as statistically significant. Statistical analyses were performed using the SPSS version 18 (IBM, Armonk, NY, USA).

RESULTS

During the study period, 681 patients had DSA, of whom 400 had their first angiogram for PAD, naive for any history of limb revascularisation. Among them, 13 patients were excluded because the renal arteries were not correctly opacified. These patients did not differ regarding age, sex, and renal function when compared with the 387 patients who constituted the study population (data not shown). The characteristics of the study population are shown in Table 1. These patients were mostly elderly males, with high rates of smoking and hypertension history. Of note, two thirds had CLI.

Table 1. Demographic, clinical, biological, and medication data in the whole population and according to presence of renal artery stenosis (RAS).

Variables	Whole cohort (n = 387)	RAS Absent (n = 330; 85%)	Present (n = 57; 15%)	p
Demographic and clinical data				
Age (y)	68.2 ± 12.2	67.5 ± 12.6	72.4 ± 8.9	.004
Male	302 (78.0)	264 (80.0)	38 (66.7)	.058
Mean ± SD BMI (kg/m^2)	25.2 ± 4.6	25.2 ± 4.8	25.3 ± 3.9	.892
Smokers	247 (63.8)	220 (66.7)	27 (47.4)	.012
Diabetes	82 (21.1)	63 (19.1)	19 (33.3)	.024
Hyperlipidemia	169 (43.7)	146 (44.2)	23 (40.4)	.584
Hypertension	246 (63.6)	203 (61.5)	43 (75.4)	.044
Heart failure	68 (17.6)	53 (16.1)	15 (26.3)	.060
Coronary artery disease	134 (34.6)	113 (34.2)	21 (36.8)	.737
Cerebrovascular disease	51 (13.2)	40 (12.1)	11 (19.3)	.139
Dialysis	14 (3.6)	11 (3.3)	3 (5.3)	.388
Critical limb ischaemia	254 (65.6)	215 (65.2)	39 (68.4)	.601
PAD distribution				
Isolated proximal	133 (37.2)	123 (40.9)	10 (17.5)	<.001
Isolated distal	44 (12.3)	40 (13.3)	4 (7.0)	
Both localisations	181 (50.5)	138 (45.8)	43 (75.4)	
Renal failure	47 (23.5)	37 (22.4)	10 (28.6)	.436
Medication				
ARB	149 (38.7)	118 (35.8)	31 (54.4)	.008
Beta-blocker	75 (19.6)	59 (17.9)	16 (28.6)	.081
Biological data (mean ± SD)				
Hemoglobin (g/L)	12.6 ± 2	12.7 ± 2.1	12.5 ± 1.8	.610
Hematocrit (%)	37.7 ± 5.8	37.8 ± 6	37.8 ± 5.3	.964
Creatinine (mmol/L)	102.1 ± 93.3	96.4 ± 83.8	122.9 ± 107.3	.038
GFR (mL/min/1.73 m^2)	87.1 ± 53.5	91.1 ± 56.2	65.6 ± 27.1	.001

Note. Data are *n* (%) unless otherwise indicated. BMI = body mass index; PAD = peripheral artery disease; ARB = angiotensin receptor blocker; GFR = glomerular filtration rate.

RAS was detected in 57 patients (14%). Comparison of these patients with the remaining 330 patients without RAS is shown in Table 1. Overall, patients with RAS were older, with higher rates of hypertension, diabetes, and heart failure, and lower rates of smoking than those without RAS. CKD rates were not higher in patients with RAS, but they had significantly lower GFRs. CLI was similarly distributed in patients with and without RAS. Patients with RAS presented more frequently with diffuse PAD. In multivariate analysis (Table 2), only two variables were significantly associated with RAS: diffuse PAD and (lower) GFR.

The follow-up duration was 62 ± 47 months (range 21 days−14 years). The follow-up index was 0.47 ± 0.35, with no significant difference between the two groups (p = .10). The event rates of different outcomes during follow-up are shown in Fig. 1. Fig. 2 shows the Kaplan−Meier survival curves for the primary and secondary outcomes. No significant difference was found for any outcome in patients with or without RAS.

In multivariate analysis, only a history of cardiovascular disease, smoking, and the presence of CLI were statistically significantly associated with the primary outcome (Table 3). Table 4 presents the multivariate model looking at variables associated with death during follow-up. Age, insulin treated diabetes, and the presence of CLI at baseline were independent predictors of mortality. Regarding the other secondary outcome (death, myocardial infarction, stroke, or coronary or carotid revascularisation), insulin treated diabetes, and CLI at baseline were the only predictors (data not shown). In none of the three models was the presence of RAS significantly associated with outcome.

Table 2. Predictive factors for the presence of renal artery stenosis in patients with peripheral artery disease (PAD): final multivariate model.

Variables		OR	95% CI	p
GFR (30 mL/min/1.73−m^2)		0.550	0.410−0.751	<.001
PAD	BK-PAD (ref.)	1	1.000−1.000	−
	AK-PAD	1.132	0.329−3.892	.844
	Diffuse PAD	3.501	1.163−10.537	.026

Note. OR = odds ratio; GFR = glomerular filtration rate; CI = confidence interval; AK = above knee; BK = below knee.

DISCUSSION

In this longitudinal study assessing the prognostic significance of RAS in patients with PAD, a high prevalence (14%) of RAS was found. However, there was no difference in outcome for patients with PAD according to the presence or absence of RAS.

The prevalence of RAS in the present series of patients with PAD is comparable with those in other similar series using the same imaging method, as summarised in Table 5.[7−14] Differences between series may be related to

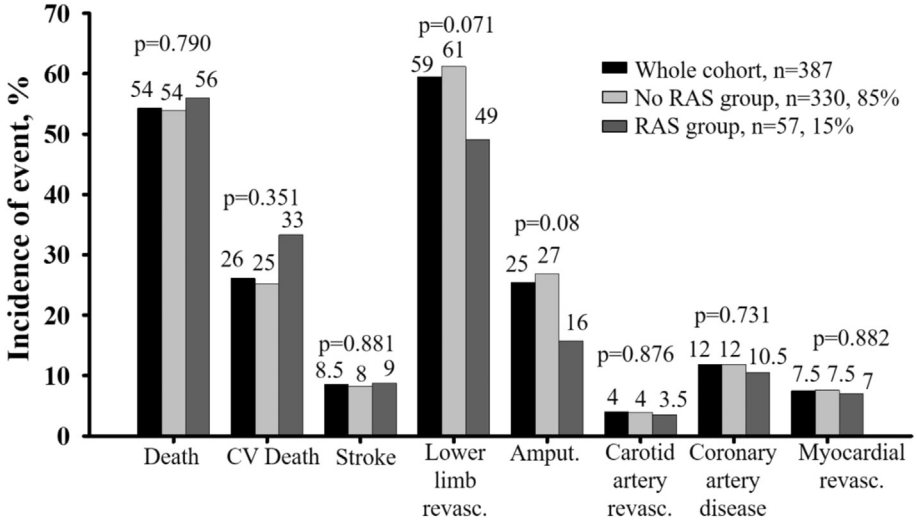

Figure 1. Prevalence of event in the whole cohort and according to the presence of renal artery stenosis (RAS). *Note*. CV = cardiovascular.

different selection criteria, RAS definition, and patient characteristics.

The lack of association between prevalent RAS and poorer outcome in patients with PAD is counterintuitive, as patients with multifocal atherosclerotic disease are usually at increased risk of cardiovascular events and death. For instance, in the REACH registry, patients with PAD who also had coronary or cerebrovascular disease presented higher

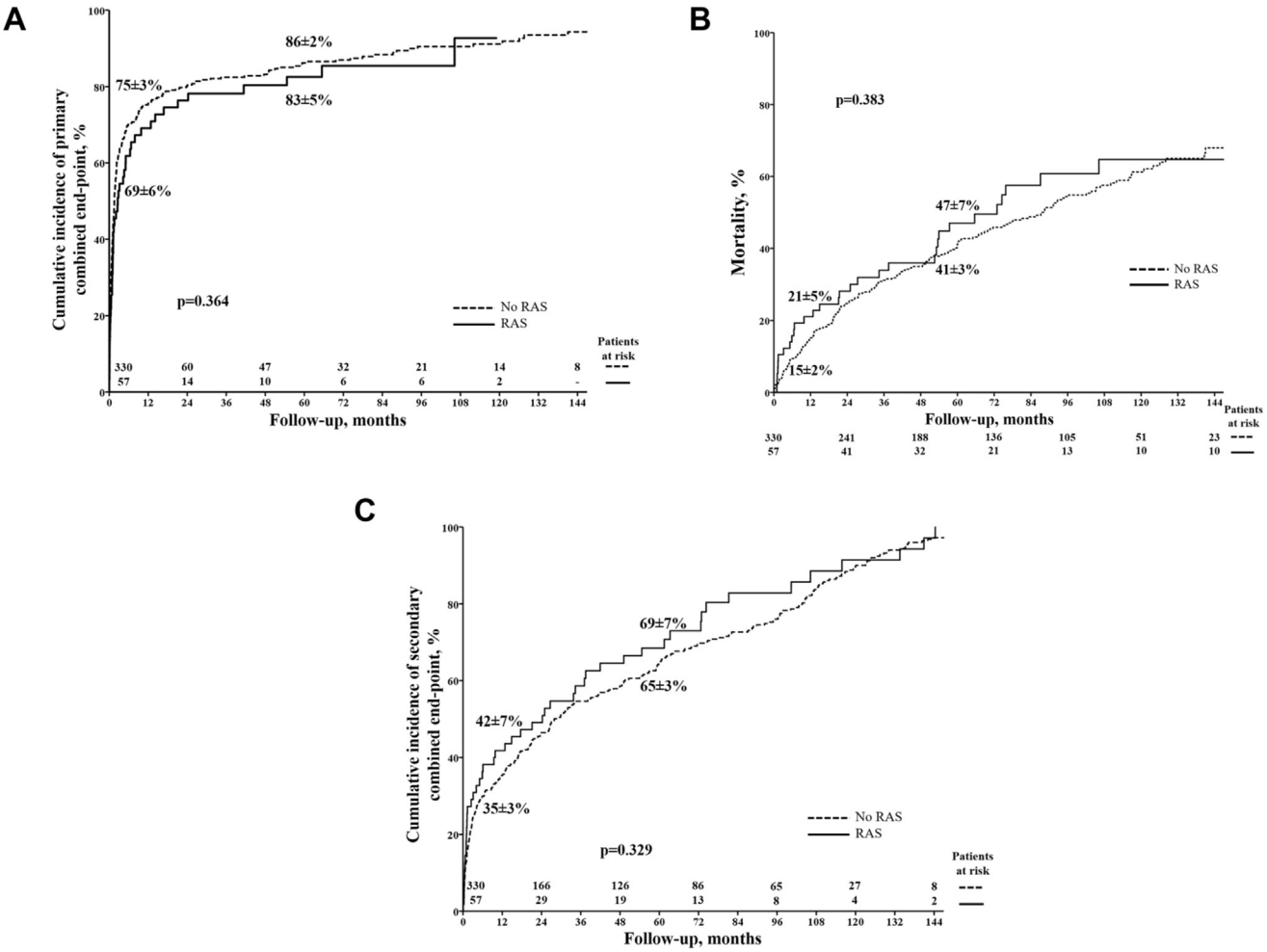

Figure 2. Kaplan—Meier analysis stratified according to the presence of renal artery stenosis (RAS). Cumulative incidence of primary combined endpoint, including (A) occurrence of death, amputation, or lower limb revascularisation, (B) mortality, and (C) cumulative incidence of secondary combined endpoint, including cardiovascular event only (i.e., cardiovascular death, myocardial infarction, stroke or coronary or carotid revascularisation) are shown.

Table 3. Predictive model for composite endpoint: death, amputation, and lower limb revascularisation.

Variable	HR	95% CI	*p*
Male sex	1.081	0.793—1.473	.622
Age (per year)	1.002	0.991—1.013	.724
History of smoking	1.346	1.006—1.801	.045
Hypertension	1.192	0.944—1.504	.140
Coronary or cerebrovascular disease	1.286	1.027—1.611	.028
Insulin treatment	0.958	0.692—1.326	.796
CLI (vs. claudication)	1.658	1.293—2.128	<.001
GFR (30 mL/min/1.73—m^2)	1.005	0.956—1.057	.837
Renal artery stenosis	0.796	0.576—1.100	.167

Note. HR = hazard ratio; CI = confidence interval; CLI = critical limb ischaemia; GFR = glomerular filtration rate.

Table 4. Predictive model for death.

Variable	HR	95% CI	*p*
Male sex	1.211	0.841—1.743	.303
Age (per year)	1.040	1.025—1.055	<.001
History of smoking	1.311	0.939—1.829	.111
Hypertension	1.072	0.784—1.466	.664
Coronary or cerebrovascular disease	1.281	0.966—1.698	.086
Insulin treatment	1.642	1.142—2.359	.007
CLI (vs. claudication)	1.743	1.249—2.432	.001
GFR (30 mL/min/1.73—m^2)	0.987	0.922—1.056	.694
Renal artery stenosis	0.871	0.587—1.294	.495

Note. HR = hazard ratio; CI = confidence interval; CLI = critical limb ischaemia; GFR = glomerular filtration rate.

rates of cardiovascular events than their counterparts without polyvascular disease.[19] The present findings can be partially explained by the fact that although older, patients with PAD plus RAS presented similar rates of clinical coronary and cerebrovascular disease, which are the drivers for increased cerebrovascular disease events.

The present findings contradict those reported from another, similar sized series of patients with PAD with comparable mean age (70 years vs. 68 years in the present study) but poorer renal function (mean GFR 64 mL/minute vs. 89 mL/minute in the present study).[11] In that study, which had a shorter follow-up of 15 months, the event rates (death cardiovascular rates and renal replacement therapy) were higher in patients with >60% RAS, detected with the same imaging method. As well as the disparities reported above, another major difference is that almost two thirds of the patients in the present study had CLI versus <12% in the other series, and the patients in the present study had more advanced stages of PAD. In turn, it is unlikely that the less stringent stenosis criteria (≥50%) in the present study would explain these contradictory results, as the authors of the other study also found an increased risk, at a lower level, for patients with 30—60% RAS.[11]

In another series of patients with unknown PAD severity (less than half were revascularised), >50% RAS was associated with increased risk of death at 5 and 10 years.[14]

It is noteworthy that another study at Dupuytren University Hospital gave similar negative results in a series of patients with severe coronary artery disease undergoing coronary bypass grafting.[20] In those patients, 10% presented with RAS detected by duplex ultrasonography, but this was not associated with outcome after a shorter follow-up duration of almost 1 year.

The present study presents several limitations. First, no specific software was used to measure the exact degree of RAS; instead, the semiquantitative "eye-ball" method was used. However, the double reading was performed by experts in vascular medicine, familiar with reading the angiographic images that were performed during that period. Also, the assessors were blinded to patient clinical data and outcomes. Second, the study was based on DSA. This imaging method has mostly been replaced by computed tomography angiography, which provides multiplanar imaging of renal arteries. Third, renal function was not systematically assessed during follow-up, so it was not possible to determine whether RAS could have any influence on renal outcome. In one study, no renal replacement therapy was required in 126 patients with PAD who had >50% RAS during follow-up,[9] while in another series with

Table 5. Prevalence of renal artery stenosis (RAS) in patients with PAD undergoing angiography: literature review.

First author	Year	*n*	Consecutive?	Age (y)	Males (%)	RAS degree (%)	RAS prevalence (%)
Choudhri[7]	1990	100	Yes	67	70	>50	42
Missouris[8]	1994	127	Yes	70	60	>50	16
Leertower[9]	2001	386	Yes but exclusion of poor imaging	68	66	>50	33
						>60	23
						>70	17
						>80	13
						>90	7
Ahmed[10]	2005	212	Yes	62	86	>50	7
Amighi[11]	2010	487	Yes but exclusion of 85 patients without renal angiography	71	58	>60	16
Endo[12]	2010	410	Not applicable (only patients who had endovascular therapy)	71	83	>50	23
						>75	11
Ozkan[13]	2009	629	Yes but exclusion of poor imaging	61	86	>60	10
Mui[14]	2011	499	Yes (one excluded for poor imaging)	66	69	>50	26
Present study	2016	400	Yes but exclusion for poor imaging	68	78	>50	14

serial assessment of GFR, renal function altered more significantly in the >60% RAS group.[11] Finally, a larger sample size may be able to detect trivial statistically significant differences between the two groups. An *a posteriori* calculation according to the 3% difference in 5 year rates of primary endpoint between patients with versus those without RAS would have required the inclusion of 2350 patients (with a power of 80%). Such a large cohort for a trivial difference would not be clinically meaningful. Alternatively, with the sample size of the present cohort, the difference in primary and secondary endpoints between the two groups should have been 17% and 20.5%, respectively, to reach 80% power with a p-value $< .05$.

Further studies carried out during earlier stages of PAD are warranted, to assess whether RAS occurring during PAD development is prognostic.

From a clinical perspective, the present study confirms that the incidental finding of RAS when imaging for PAD is frequent. However, clinicians taking care of patients with PAD should not consider the coexistence of RAS to be associated with a poorer cardiovascular prognosis as in multifocal atherosclerosis, as is suggested for other localisations of atherosclerosis (e.g., coronary artery disease).

Conclusions

In the present study, 14% of patients hospitalised for PAD presented concomitant RAS, especially in those with diffuse PAD and lower GFRs. However, in the study, the coexistence of these lesions in patients with PAD was not found to be associated with poorer prognosis, neither with regard to their lower extremities nor for general outcomes. Larger, multicentre studies are necessary to draw definite conclusions.

CONFLICT OF INTEREST

None.

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