Renal artery stenosis in patients with coronary artery disease

Tadeusz Przewłocki[1], Anna Kabłak-Ziembicka[1], Wiesława Tracz[1], Artur Kozanecki[1], Grzegorz Kopeć[1], Paweł Rubiś[1], Magdalena Kostkiewicz[1], Agnieszka Rosławiecka[1], Daniel Rzeźnik[1], Tomasz Stompór[2]

[1] Department of Cardiac and Vascular Diseases, Institute of Cardiology, *Collegium Medicum*, Jagiellonian University, The John Paul II Hospital, Krakow, Poland

[2] Chair and Department of Nephrology, University Hospital, Krakow, Poland

Abstract

Background: Renal dysfunction is an important factor of cardiovascular risk. Renal artery stenosis (RAS) is a potential cause of secondary hypertension and by renal ischemia may lead to progressive renal insuficiency. In RAS patients a significant increase in prevalence of coronary artery disease (CAD) as well as revascularisation rate and mortality rate was observed.

Aim: To determine the prevalence of RAS in patients with suspected CAD.

Methods: The study group consisted of 1036 consecutive patients (700 men; 67.6%) in the mean age of 62.1±9.7 (25–85) years admitted to coronary angiography. Simultaneously renal angiography was performed in all patients.

Results: Stenosis ≥50% in at least one main branch of coronary artery was found in 633 (66.1%) patients. The proportion of patients with one, two or three vessel CAD was respectively 291 (46%), 169 (26%) and 173 (27.3%). Non-significant coronary lesions <50% were found in 108 (10.4%) patients, whereas in 295 (28.5%) patients no angiographic evidence of CAD was documented. In the whole group of patients RAS was found in 339 (32.7%) of patients – 124 (12%) had bilateral lesions. RAS prevalence in patients with CAD was 38.3% (284/741) and its frequency increased with severity of CAD: from 25% (27/108) in patients with insignificant coronary lesions up to 36.4% (106/291), 40.2% (68/169) and 48% (83/173) in 1, 2, and 3-vessel disease, respectively (p <0.001). RAS prevalence in patients with normal coronary arteries was 18.6% (55/295). RAS <30% was detected in 194 (18,7%) patients; RAS between 30-49% in 81 patients (8.7%); RAS 50–69% in 38 patients (3.7%) and RAS ≥70% in the remaining 26 patients (2.5%). RAS ≥50% was noted in 8 (2.7%) patients without coronary lesions; in 5 (4.6%) with insignificant coronary artery atherosclerosis and 51 (8%) with coronary artery stenosis >50% (p=0.0008). Stepwise regression analysis identified 4 independent predictors of RAS ≥50%: CAD severity (p=0.014), serum creatinine concentration (p <0.001), cigarette smoking (p=0.02) and stenosis of aortic arch branches (p <0.001).

Conclusions: RAS is a frequent finding in patients with suspected CAD. CAD severity, number of involved aortic arch arteries, cigarette smoking and serum creatinine are independent RAS predictors.

Key words: renal artery stenosis, coronary artery disease, predictors

Kardiol Pol 2008; 66: 856-862

Introduction

Renal artery stenosis (RAS) worsens the course of coronary artery disease (CAD), leading at the same time to more frequent episodes of myocardial infarction, coronary revascularisation and, worst of all, to increased mortality [1-3]. It has been known for a long time that RAS is one of the mechanisms contributing to development of systemic hypertension and chronic renal failure [4-6]. In recent years, a growing number of patients referred for coronary angiography and revascularisation have advanced hypertension and renal failure. Therefore, identification of patients at high risk of RAS could be of great clinical relevance and affect therapeutic decisions [7-10]. Different estimations of RAS prevalence can be found, depending on the age and characteristics of populations studied, concomitant risk factors and social and environmental determinants [11-14].

The aim of our study was to assess the prevalence of RAS among patients with suspected CAD referred for invasive coronary angiography and attempt to identify predictors of RAS.

Adres do korespondencji:

Tadeusz Przewłocki MD, PhD, Krakowski Szpital Specjalistyczny im. Jana Pawła II, ul. Prądnicka 80, 31-202 Kraków, tel.: +48 12 614 20 00, fax: +48 12 614 22 66, e-mail: tadeuszprzewlocki@op.pl

Received: 12 February 2008. **Accepted:** 08 May 2008.

This study received financial support from an educational grant of Ministerstwo Nauki i Szkolnictwa Wyższego (KBN), project number 3PO5B10325

Methods

Study group

The study involved 1036 consecutive patients (700 males; 67.6%) at the mean age of 62.1±9.7 (25–85) years, admitted to our Department over a period of 12 months with suspected CAD to be confirmed with coronary angiography.

Coronary angiography

All patients had coronary angiography carried out using the Seldinger technique and femoral or radial artery according to well established practices and using Axiom Artis DFC or Coroscop systems, Siemens, with quantitative assessment of coronary artery stenosis using the tool Quantcor QCA V2.0 [15]. The patients were divided into three groups with respect to coronary angiography results: patients without apparent atherosclerotic lesions in the main coronary branches, patients with distinct, but insignificant stenotic lesions (≤50% reduction in lumen diameter) and patients with >50% stenosis in the main coronary arteries. Depending on the number and location of these lesions, patients with >50% stenotic plaques were classified as having single-, two- or three-vessel disease.

Angiography of renal arteries

All patients had renal angiography performed together with coronary angiography using the same equipment and software. In the majority of patients selective angiography of renal arteries was done employing a 6 French right Judkins catheter. When ventriculography was performed, angiography of renal arteries was done unselectively using a pigtail catheter. In order to determine the percentage of lumen reduction quantitative angiography was used; lumen reduction <30% was defined as minimum, 30-49% as mild, 50-69% as moderate, and ≥70% as severe.

Risk factors of atherosclerosis

The following risk factors of atherosclerosis were assessed in all patients: hypertension, hyperlipidaemia, diabetes, cigarette smoking, gender, age, lipid profile, and serum creatinine, glucose and high sensitivity C-reactive protein (hs–CRP) blevels.

Statistical methods

The study parameters are presented as means and standard deviations for continuous variables and as numbers and percentages for discrete variables. Differences in distribution of values and frequency of evaluated parameters between the study groups were assessed with Chi-square test. The differences in prevalence of RAS in relation to the severity of coronary artery disease were analysed using analysis of variance (ANOVA).

Independent predictors of renal artery atherosclerosis and RAS ≥50% were sought using multivariate step-wise regression. The analysis involved 17 clinical and angiographic variables, i.e. age, sex, cigarette smoking, hypertension, diabetes, body mass index (BMI), history of myocardial infarction, past stroke, severity of CAD, ≥50% stenosis of aortic arch branch, hyperlipidaemia, concentration of total cholesterol, LDL and HDL cholesterol, triglycerides, creatinine and hs-CRP.

Statistical analyses were performed using *Statistica 5.5* software. The statistical significance level of $p < 0.05$ was used.

Results

Coronary angiography revealed stenosis of ≥50% in at least one of the main coronary vessels in 633 (61.1%) patients, including 291 (46%) cases with single-vessel CAD, 169 (26.7%) with two-vessel CAD and 173 (27.3%) with three-vessel CAD. A group of 108 (10.4%) patients had atherosclerotic lesions with <50% stenosis and coronary angiography was negative in 295 (28.5%) patients. Clinical characteristics of patients are shown in Tables I and II.

Patients without CAD, those with minor luminal irregularities and those with significant stenoses differed considerably from each other with respect to age, cardiovascular risk factor burden, extra-cardiac atherosclerotic lesions and serum creatinine, hs-CRP and lipid fraction concentrations (Table I). However, the differences between subgroups of patients with significant coronary lesions were slight and significant only for age and creatinine concentration (Table II).

In 339 of 1036 patients (32.7%) atherosclerotic lesions in the renal arteries were detected, including bilateral changes in 124 (12%) subjects. The distribution of severity of these lesions was as follows: stenosis <30% – 194 (57%) patients; 30 to 49% – 81 (24%); 50 to 69% – 38 (11.2%); and ≥70% – 26 (7.6%) patients. Thus, among 339 patients with RAS moderate and severe lesions (≥50% stenosis) were observed in 18.8%, that is in every fifth patient.

In patients with normal coronary arteries, prevalence of RAS was 18.6% (55/295 patients). In patients with CAD prevalence of RAS was 38.3% (284/741), and depended upon the severity of CAD, reaching 25% (27/108) in subjects with minor luminal changes and 36.4% (106/291), 40.2% (68/169) and 48% (83/173) in patients with 1-, 2-, and 3-vessel disease, respectively ($p < 0.001$) (Table III).

Renal artery stenosis <30% was found in 194 (18.7%) patients; stenosis of 30-49% in 81 (8.7%); of 50-69% in 38 (3.7%); and ≥70% in 26 (2.5%) patients respectively.

Renal artery stenosis ≥50% was found in 8 (2.7%) patients without CAD on angiography; in 5 (4.6%) patients with minor luminal irregularities in coronary arteries and in 51 (8%) with ≥50% coronary artery stenosis ($p=0.0008$). These data are summarised in Table III. Additionally, Table III shows the prevalence of lesions classified according to previously defined RAS ranges in patients with CAD of various severity and in those without CAD.

Table I. Clinical characteristics of patients in relation to the severity of atherosclerotic CAD

	Coronary angiography			p
	No coronary lesions N=295	Coronary lesions stenosis <50% N=108	Coronary lesions stenosis >50% N=633	
Age [years±SD]	60.0±9.6	63.2±9.3	62.9±9.6	<0.001
Males [n]	136 (46.1%)	72 (66.7%)	492 (77.7%)	<0.001
BMI [kg/m²±SD]	27.6±4.8	28.4±4.3	28.5±4.1	0.122
Hypertension [n]	214 (72.5%)	92 (85.2%)	526 (83.1%)	<0.001
Diabetes mellitus [n]	29 (9.8%)	24 (22.2%)	136 (21.4%)	<0.001
Hyperlipidaemia [n]	228 (77.2%)	97 (89.8%)	574 (91.0%)	<0.001
Smoking [n]	116 (39.3%)	64 (59.3%)	386 (61.0%)	<0.001
Myocardial infarction [n]	0 (0%)	16 (14.8%)	296 (47.0%)	<0.001
Stroke [n]	15 (5.1%)	9 (8.3%)	44 (7.0%)	0.876
Carotid artery stenosis ≥50%	0 (0%)	7 (6.5%)	73 (11.5%)	<0.001
Creatinine [µmol/l±SD]	79.9±20.5	87.3±26.4	90.1±32	<0.001
hs-CRP [mg/dl±SD]	3.1±4.1	3.0±3.3	4.4±7.7	0.027
HDL [mmol/l±SD]	1.34±0.38	1.23±0.33	1.18±0.39	<0.001
LDL [mmol/l±SD]	2.99±0.88	3.06±1.05	3.08±0.95	0.426
Triglycerides [mmol/l±SD]	1.32±0.68	1.68±1.18	1.66±1.01	<0.001

Table II. Clinical characteristics of patients with coronary artery stenosis of >50% in relation to the number of main coronary branches involved

	Coronary artery stenosis of >50%			p
	1-vessel CAD N=291	2-vessel CAD N=169	3-vessel CAD N=173	
Age [years±SD]	62.1±9.4	62.4±10	64.5±9.5	**0.028**
Males [n]	215 (73.9%)	135 (79.8%)	142 (82.1%)	0.090
BMI [kg/m²±SD]	28.1±6.1	28.5±5.6	29.1±5.8	0.356
Hypertension [n]	242 (83.2%)	137 (81.1%)	148 (85.5%)	0.540
Diabetes mellitus [n]	52 (17.9%)	39 (23.1%)	45 (26.0%)	0.100
Hyperlipidaemia [n]	259 (89.3%)	157 (92.3%)	158 (91.3%)	0.424
Smoking [n]	180 (61.9%)	98 (58.0%)	108 (62.4%)	0.645
Myocardial infarction [n]	122 (42.5%)	179 (47.3%)	103 (60.0%)	**0.003**
Stroke [n]	15 (5.1%)	15 (8.9%)	14 (8.1%)	0.251
Creatinine [µmol/l±SD]	86.4±24.7	90.3±28.1	96.1±43.4	**0.006**
hs-CRP [mg/dl±SD]	4.1±7.3	5.2±11.4	4.8±7.8	0.407
HDL [mmol/l±SD]	1.19±0.37	1.15±0.35	1.19±0.56	0.521
LDL [mmol/l±SD]	3.01±0.91	3.16±0.98	3.11±0.98	0.240
Triglycerides [mmol/l±SD]	1.61±0.94	1.77±1.22	1.64±0.89	0.248

In patients with significant coronary artery lesions the prevalence of RAS ≥50% increased along with severity of CAD, reaching 6.3% in 1-vessel CAD, 8.3% in 2-vessel CAD and 10.4% in 3-vessel CAD (p=0.048).

Univariate analysis showed that RAS was more prevalent in females than males, in hypertensive patients than in those without hypertension and in patients with hyperlipidaemia compared to those without (Figure 1). A weak correlation between prevalence of RAS and patients' age was found (r=0.227, p <0.001).

Multivariate step-wise regression analysis identified 8 independent predictors of renal artery atherosclerosis (Table IV), including the presence of ≥50% stenosis of at least one of the major aortic arch branches, severe CAD,

Table III. Prevalence and severity of renal artery disease in relation to the severity of CAD

	Normal renal arteries	RAS <30%	RAS 30-49%	RAS 50-69%	RAS ≥70%	p
No CAD; N=295	240 (81.4%)	36 (12.2%)	11 (3.7%)	7 (2.4%)	1 (0.3%)	0.041
Coronary arteries with minor luminal irregularities <50%; N=108	81 (75.0%)	15 (13.9%)	7 (6.5%)	1 (0.9%)	4 (3.7%)	
Stenosis of coronary arteries of ≥50%; N=633	376 (59.4%)	143 (22.6%)	63 (10.0%)	30 (4.7%)	21 (3.3%)	0.023
Including:						
1-vessel CAD; N=291	185 (63.6%)	63 (21.6%)	24 (8.2%)	**11 (3.8%)**	**8 (2.7%)**	0.051
2-vessel CAD; N=169	101 (59.8%)	35 (20.7%)	19 (11.2%)	**8 (4.7%)**	**6 (3.6%)**	
3-vessel CAD; N=173	90 (52.0%)	45 (26.0%)	20 (11.6%)	**11 (6.4%)**	**7 (4.0%)**	0.0571

female gender, age, diabetes mellitus, hypertension, serum creatinine and total cholesterol levels.

Independent predictors of RAS ≥50% included the presence of ≥50% stenosis of at least one of the major aortic arch branches, severe CAD, female gender, creatinine level and smoking (Table V).

Discussion

Our study shows that RAS is a relatively common finding in patients referred for diagnostic coronary angiography, being found in 1/3 of them. Prevalence of RAS significantly rises along with severity of atherosclerotic CAD, from 18% in patients without apparent coronary atherosclerosis to 48% in 3-vessel CAD. However, the prevalence of RAS ≥50% is considerably lower and ranges from 2.7% in patients without atherosclerotic lesions in coronary arteries to 8% in subjects with confirmed coronary artery stenosis of >50% and 10% in subjects with 3-vessel CAD. In total, RAS ≥50%, which may possibly significantly affect renal performance and induce hypertension, occurs in every fifth patient with atherosclerotic lesions in the renal arteries, which is relatively rare.

Table IV. Independent predictors of atherosclerotic renal artery disease (regardless of its severity)

Predictors	Beta	SE	p
Intercept			**<0.001**
Aortic arch branch stenosis ≥50%	0.143	0.031	**<0.001**
Severity of CAD	0.190	0.032	**<0.001**
Male gender	–0.120	0.033	**<0.001**
Age	0.165	0.032	**<0.001**
Diabetes mellitus	0.080	0.031	**0.009**
Hypertension	0.056	0.031	**0.067**
Creatinine level	0.097	0.033	**0.003**
Cholesterol level	0.097	0.031	**0.002**

The prevalence of renal artery atherosclerosis evaluated during diagnostic angiography of different vascular beds is reported to range from 7 to 70% [8, 11, 16–18]. The prevalence of RAS in patients undergoing cardiac catheterisation due to suspected CAD ranges from 11.3 to 39%, whereas of RAS with stenosis of >50% – from 6.3 to 28% [19-25]. Rates found in our study remain within these ranges, indicating that our population was

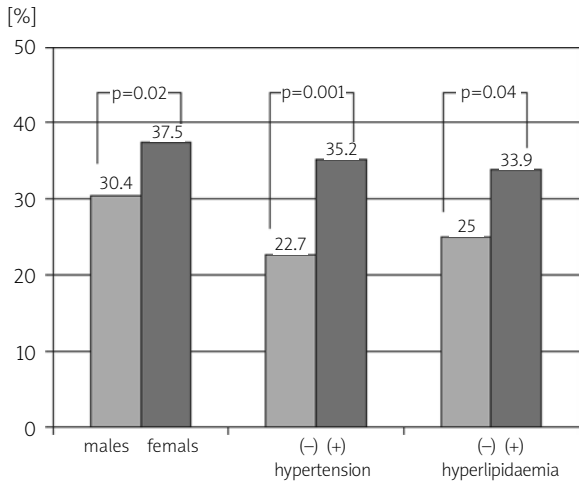

Figure 1. Prevalence of renal atherosclerosis in different subgroups

Table V. Independent risk factors of renal artery stenosis of ≥50%

Risk factors	Beta	SE	p
Intercept			**0.045**
Aortic arch branch stenosis ≥50%	0.163	0.032	**<0.001**
Severity of CAD	0.079	0.032	**0.014**
Male gender	–0.105	0.034	**0.002**
Smoking	0.074	0.032	**0.020**
Creatinine level	0.250	0.032	**<0.001**

similar to those studied elsewhere with respect to the age and distribution of risk factors.

Interestingly, RAS rate increased with the progressing severity of CAD. A similar observation was reported by Weber-Mzell et al., in a significantly smaller number of patients [20]. Searching for independent risk factors of significant (≥50%) RAS we identified as significant the following ones: stenosis of major aortic arch branch, serum creatinine, female gender, severity of CAD and smoking. Severity of CAD was also found to be a significant independent risk factor in studies of Weber-Mzell et al. [20] and Harding et al. [22]. Coronary artery disease being a predictor of RAS was reported by Crowley et al. [8], Alhaddad et al. [24], Park et al. [25] and Cohen et al. [26]. The presence of peripheral artery disease, female gender and serum creatinine as independent risk factors was confirmed by Crowley et al. [8], Aqel et al. [21], Harding et al. [22], Buller et al. [23], Alhaddad et al. [24], and Park et al. [25]. In our study group, age and hypertension were identified as the risk factors of atherosclerosis but not RAS ≥50%, which is similar to findings reported by others.

Routine renal artery angiography for detection of RAS, performed during vascular procedures aimed at different vascular targets, has not gained widespread acceptance [11, 27]. Therefore, stratifying the risk of coexisting significant RAS may be relevant in patients undergoing angiography due to suspected stenotic lesions in other vascular beds, e.g. coronary, lower limb or aortic arch branch angiography. If clinically indicated, RAS can be easily confirmed or excluded using the same diagnostic catheters and injecting only a slightly higher amount of contrast agent. Thus a number of investigators have attempted to develop RAS predictive models based on the simplest possible clinical parameters [26, 28, 29]. Although these models have moderate sensitivity and specificity for detecting RAS of ≥50%, they may be useful for identifying patients who should undergo renal angiography during a planned vascular examination. Renal angiography seems to be justified in patients with a high burden of risk factors [28, 29]. However, it should be emphasised that diagnosis of RAS ≥50% alone is not an automatic indication for therapeutic intervention, which may be performed only after a causal association of RAS and renal failure and/or hypertension is proved [11, 30-34].

Conclusions

Renal artery stenosis is common in patients with suspected CAD, being found in 1/3 of them, whereas moderate-to-severe stenosis is detected in 8% of patients with significant CAD. Prevalence of RAS increases with severity of CAD. The independent predictors of RAS are severity of CAD, stenosis of the aortic arch branches, smoking and serum creatinine.

References

1. Conlon PJ, Little MA, Pieper K, et al. Severity of renal vascular disease predicts mortality in patients undergoing coronary angiography. *Kidney Int* 2001; 60: 1490-7.
2. Conlon PJ, Athirakul K, Kovalik E, et al. Survival in renal vascular disease. *J Am Soc Nephrol* 1998; 9: 252–6.
3. Edwards MS, Craven TE, Burke GL, et al. Renovascular disease and the risk of adverse coronary events in the elderly. *Arch Intern Med* 2005; 165: 207-13.
4. Garovic VD, Textor SC. Renovascular hypertension and ischemic nephropathy. *Circulation* 2005; 112: 1362-74.
5. Tobe SW, Burgess E, Lebel M. Atherosclerotic renovascular disease. *Can J Cardiol* 2006; 22: 623-8.
6. Hollenberg NK. Renal hemodynamics in essential and renovascular hypertension. Influence of captopril. *Am J Med* 1984; 76: 22-8.
7. Dudek D, Chyrchel B, Siudak Z, et al. Renal insufficiency increases mortality in acute coronary syndromes regardless of TIMI risk score. *Kardiol Pol* 2008; 66: 28-34.
8. Crowley JJ, Santos RM, Peter RH, et al. Progression of renal artery stenosis in patients undergoing cardiac catheterization. *Am Heart J* 1998; 136: 913-8.
9. Roussos L, Christensson A, Thompson O. A study on the outcome of percutaneous transluminal renal angioplasty in patients with renal failure. *Nephron Clin Pract* 2006; 104: c132-42.
10. Bates MC, Campbell JE, Stone PA, et al. Factors affecting long-term survival following renal artery stenting. *Catheter Cardiovasc Interv* 2007; 69: 1037-43.
11. Androes MP, Langan EM 3rd, Kalbaugh CA, et al. Is incidental renal arteriography justified in a population of patients with symptomatic peripheral arterial disease? *Vasc Endovascular Surg* 2007; 41: 106-10.
12. Esteban C, Presas A, Ara J, et al. Prevalence of renal artery stenosis in patients with critical limb ischemia. *Med Clin* (*Barc*) 2007; 128: 561-4.
13. de Silva R, Loh H, Rigby AS, et al. Epidemiology, associated factors, and prognostic outcomes of renal artery stenosis in chronic heart failure assessed by magnetic resonance angiography. *Am J Cardiol* 2007; 100: 273-9.
14. Kawarada O, Yokoi Y, Morioka N, et al. Renal artery stenosis in cardio-and cerebrovascular disease: renal duplex ultrasonography as an initial screening examination. *Circ J* 2007; 71: 1942-7.
15. ACC/AHA/SCAI 2005 Guideline Update for Percutaneous Coronary Intervention. A Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (ACC/AHA/SCAI Writing Committee to Update the 2001 Guidelines for Percutaneous Coronary Intervention). *J Am Coll Cardiol* 2005; 1: 3-121.
16. Wachtell K, Ibsen H, Olsen MH, et al. Prevalence of renal artery stenosis in patients with peripheral vascular disease and hypertension. *J Hum Hypertens* 1996; 10: 83-5.
17. Swartbol P, Pärsson H, Thorvinger B, et al. To what extent does peripheral vascular disease and hypertension predict renal artery stenosis? *Int Angiol* 1994; 13: 109-114.
18. Choudhri AH, Cleland JG, Rowlands PC, et al. Unsuspected renal artery stenosis in peripheral vascular disease. *BMJ* 1990; 301: 1197-8.
19. Rihal CS, Textor SC, Breen JF, et al. Incidental renal artery stenosis among a prospective cohort of hypertensive patients undergoing coronary angiography. *Mayo Clin Proc* 2002; 77: 309-16.
20. Weber-Mzell D, Kotanko P, Schumacher M, et al. Coronary anatomy predicts presence or absence of renal artery stenosis. *Eur Heart J* 2002; 23: 1684-91.

21. Aqel RA, Zoghbi GJ, Baldwin SA, et al. Prevalence of renal artery stenosis in high-risk veterans referred to cardiac catheterization. *J Hypertens* 2003; 21: 1157-62.
22. Harding MB, Smith LR, Himmelstein SI, et al. Renal artery stenosis: prevalence and associated risk factors in patients undergoing routine cardiac catheterization. *J Am Soc Nephrol* 1992; 2: 1608-16.
23. Buller CE, Nogareda JG, Ramanathan K, et al. The profile of cardiac patients with renal artery stenosis. *J Am Coll Cardiol* 2004; 43: 1606-13.
24. Alhaddad IA, Blum S, Heller EN, et al. Renal artery stenosis in minority patients undergoing diagnostic cardiac catheterization: prevalence and risk factors. *J Cardiovasc Pharmacol Ther* 2001; 6: 147-53.
25. Park S, Jung JH, Seo HS, et al. The prevalence and clinical predictors of atherosclerotic renal artery stenosis in patients undergoing coronary angiography. *Heart Vessels* 2004; 19: 275-9.
26. Cohen MG, Pascua JA, Garcia-Ben M, et al. A simple prediction rule for significant renal artery stenosis in patients undergoing cardiac catheterization. *Am Heart J* 2005; 150: 1204-11.
27. Dear JW, Padfield PL, Webb DJ. New guidelines for drive-by renal arteriography may lead to an unjustifiable increase in percutaneous intervention. *Heart* 2007; 93: 1528-32.
28. Krijnen P, van Jaarsveld BC, Steyerberg EW, et al. A clinical prediction rule for renal artery stenosis. *Ann Intern Med* 1998; 129: 705-11.
29. Przewłocki T, Kabłak-Ziembicka A, Tracz W, et al. Prevalence and prediction of renal artery stenosis in patients with coronary and supraaortic artery atherosclerotic disease. *Nephrol Dial Transplant* 2008; 23: 580-5.
30. Mui KW, Woittiez AJ, Navis G. Atherosclerotic renovascular disease and renal impairment: can we predict the effect of intervention? *Curr Hypertens Rep* 2007; 9: 140-7.
31. Tagle R, Acevedo M, Xu M, et al. Use of endovascular stents in atherosclerotic renovascular stenosis: blood pressure and renal function changes in hypertensive patients. *J Clin Hypertens* 2007; 9: 608-14.
32. White CJ. Catheter-based therapy for atherosclerotic renal artery stenosis. *Circulation* 2006; 113: 1464-1673.
33. Kręcki R, Drożdż J, Krzemińska-Pakuła M. Prognostic factors in patients with advanced multi-vessel coronary artery disease. *Kardiol Pol* 2006; 64: 1179-85.
34. Ratnam LA, Nelson SR, Belli AM. Investigating progressive unexplained renal impairment and hypertension. *BMJ* 2007; 335: 1094-6.

Występowanie zwężenia tętnic nerkowych u osób z chorobą niedokrwienną serca

Tadeusz Przewłocki[1], Anna Kabłak-Ziembicka[1], Wiesława Tracz[1], Artur Kozanecki[1], Grzegorz Kopeć[1], Paweł Rubiś[1], Magdalena Kostkiewicz[1], Agnieszka Rosławiecka[1], Daniel Rzeźnik[1], Tomasz Stompór[2]

[1] Krakowski Szpital Specjalistyczny im. Jana Pawła II

[2] Katedra i Klinika Nefrologii, Szpital Uniwersytecki, Kraków

Streszczenie

Wstęp: Zwężenie tętnicy nerkowej (RAS) może być przyczyną nadciśnienia tętniczego oraz poprzez niedokrwienie prowadzić do postępującej niewydolności nerek, która jest istotnym czynnikiem ryzyka sercowo-naczyniowego. U chorych z RAS wykazano znamiennie wyższą zapadalność na chorobę wieńcową (CAD), częstość rewaskularyzacji i niższą przeżywalność.

Cel: Celem badania była ocena częstości występowania RAS u osób z CAD.

Metody: Badaną grupę stanowiło 1036 kolejnych chorych (700 mężczyzn; 67,6%) w wieku 62,1±9,7 roku (25–85 lat) przyjętych z podejrzeniem CAD. U wszystkich wykonano koronarografię oraz angiografię tętnic nerkowych.

Wyniki: Na podstawie wyniku koronarografii u 633 (61,1%) chorych stwierdzono zwężenie ≥50% w co najmniej jednej z głównych tętnic wieńcowych, w tym u odpowiednio u 291 (46%), 169 (26,7%) oraz 173 (27,3%) chorych rozpoznano 1-, 2- oraz 3-naczyniową CAD. U 108 (10,4%) chorych zmiany miażdżycowe w tętnicach wieńcowych nie przekraczały 50% redukcji światła naczynia, a u 295 (28,5%) chorych stwierdzono prawidłowe tętnice wieńcowe. W całej badanej grupie zmiany miażdżycowe w tętnicach nerkowych stwierdzono u 339 (32,7%) chorych, w tym u 124 (12%) występowały obustronnie. Natomiast wśród chorych ze zmianami miażdżycowymi w tętnicach wieńcowych częstość RAS wynosiła 38,3% (284/741) i wzrastała z zaawansowaniem CAD, wynosi 25% (27/108) u chorych z przyściennymi, i odpowiednio 36,4% (106/291), 40,2% (68/169) oraz 48% (83/173) wśród chorych z 1-, 2- oraz 3-naczyniową CAD (p <0,001). Wśród chorych z prawidłowymi tętnicami wieńcowymi częstość RAS wynosiła 18,6% (55/295). RAS <30% stwierdzono u 194 (18,7%) chorych, pomiędzy 30–49% u 81 (8,7%), pomiędzy 50–69% u 38 (3,7%) oraz ≥70% u 26 (2,5%) chorych. Częstość RAS ≥50% dotyczyła 8 (2,7%) chorych bez CAD, 5 (4,6%) chorych z przyściennymi blaszkami miażdżycowymi w tętnicach wieńcowych oraz 51 (8%) ze zwężeniami w tętnicach wieńcowych >50% (p=0,0008). W analizie metodą wieloczynnikowej regresji krokowej wykazano, że niezależnymi predyktorami RAS ≥50% był stopień zaawansowania CAD (p=0,014), stężenie kreatyniny (p <0,001), palenie papierosów (p=0,02) oraz zwężenia tętnic odchodzących od łuku aorty ≥50% (p <0,001).

Wnioski: Zwężenie tętnicy nerkowej jest częstym zjawiskiem u chorych z CAD. Niezależnymi czynnikami ryzyka występowania RAS są: stopień zaawansowania CAD, zwężenia tętnic łuku aorty, palenie papierosów i stężenie kreatyniny.

Słowa kluczowe: zwężenie tętnicy nerkowej, choroba wieńcowa, czynniki predykcyjne

Kardiol Pol 2008; 66: 856-862

Adres do korespondencji:

dr hab. n. med. Tadeusz Przewłocki, Krakowski Szpital Specjalistyczny im. Jana Pawła II, ul. Prądnicka 80, 31-202 Kraków, tel.: +48 12 614 20 00, faks: +48 12 614 22 66, e-mail: tadeuszprzewlocki@op.pl

Praca wpłynęła: 12.02.2008. **Zaakceptowana do druku:** 08.05.2008.