Original Article

The Incidence and Risk Factors of Renal Artery Stenosis in Patients with Severe Carotid Artery Stenosis

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We previously showed that renal artery stenosis (RAS) was commonly found in patients with cardiovascular disease (CVD) such as myocardial infarction, stroke, or abdominal aneurysm. The aim of the present study was to evaluate the incidence and risk factors for RAS in patients with severe carotid artery stenosis (CAS) considered to need carotid endarterectomy. From February to August 2006, 41 consecutive patients with severe CAS were admitted to the Department of Neurosurgery of the National Cardiovascular Center. Each patient was examined for renal function and urinary albumin excretion, and renal artery duplex scanning was also performed. The patients were classified into two groups according to the findings of renal Doppler sonography, 11 patients with RAS and 30 patients without RAS. We evaluated the differences in clinical findings and renal function between the groups and clarified the risk factors for RAS. In RAS patients, smoking and incidence of other CVDs were evident, and renal function was impaired significantly compared with the patients without RAS. Multivariate logistic regression showed that the presence of other CVDs, renal function, and smoking were significant clinical predictors for RAS. In patients with severe CAS, RAS was frequently detected with the same frequency as ischemic heart disease. The RAS risk factors were the presence of other CVDs, renal dysfunction, and smoking. Since RAS is an underlying cause of hypertension and renal failure, it is important to consider the presence of RAS in patients with severe CAS. (*Hypertens Res* 2007; 30: 839–844)

***Key Words*: renal artery stenosis, carotid artery stenosis, smoking, renal dysfunction, atherosclerosis**

Introduction

Renal artery stenosis (RAS) is the most common cause of secondary hypertension in patients with atherosclerosis. Atherosclerotic RAS accounts for 60% to 97% of all cases of RAS, and is also becoming recognized as an important cause of ischemic renal failure (*1*). Ischemic renal disease has been reported to be the cause of 10 to 20% of end-stage renal disease (ESRD) cases (*1–3*).

Several studies have shown that a high percentage of significant atherosclerotic RAS cases are clinically indolent. Harding *et al*. found significant RAS in 15% of patients undergoing routine cardiac catheterization (*4*). Our previous studies have shown coexisting coronary artery disease, stroke, and abdominal aortic aneurysm to be highly predictive of significant RAS (*5–7*). The clinical importance of these indolent lesions is that they may continue to progress and result in significant morbidity and mortality associated with ischemic nephropathy and ESRD (*3*).

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Received January 25, 2007; Accepted in revised form April 19, 2007.

Table 1. Baseline Characteristics of the Study Subjects

Variables	Patients with RAS (n=11)	Patients without RAS (n=30)	p value
Age (years old)	70±8	66±9	n.s.
Male gender (n (%))	10 (91)	22 (73)	n.s.
Body mass index (kg/m^2)	25.0±3.6	23.2±3.5	n.s.
Hypertension (n (%))	10 (91)	25 (83)	n.s.
Diabetes (n (%))	6 (55)	16 (53)	n.s.
Hyperlipemia (n (%))	8 (73)	18 (60)	n.s.
Coronary artery disease (n (%))	9 (82)	9 (30)	<0.005
Prior MI (n (%))	3 (27)	3 (10)	0.09
PCI (n (%))	6 (55)	4 (13)	<0.05
CABG (n (%))	1 (9)	4 (13)	n.s.
Cerebral vascular disease (n (%))	7 (64)	19 (63)	n.s.
Peripheral vascular disease (n (%))	5 (45)	3 (10)	<0.05
Current smoking (n (%))	10 (91)	16 (53)	<0.05
Systolic blood pressure (mmHg)	148±8	142±14	n.s.
Diastolic blood pressure (mmHg)	76±14	77±10	n.s.
Pulse pressure (mmHg)	72±12	65±12	n.s.
Total cholesterol (mg/dL)	157±39	172±33	n.s.
Triglycerides (mg/dL)	113±81	135±76	n.s.
HDL-cholesterol (mg/dL)	40±11	41±15	n.s.
PRA (ng/mL/h)	2.4±1.6	2.5±4.8	n.s.
LVMI (g/m^2)	105±25	109±34	n.s.
FS (%)	39±15	36±12	n.s.
CAS (unilateral/bilateral)	6/5	10/20	n.s.
CAS (with/without occlusion)	2/9	9/21	n.s.
Degree of CAS (area) (%)	92±4	90±9	n.s.
Degree of CAS (NASCET) (%)	72±10	69±13	n.s.

RAS, renal artery stenosis; MI, myocardial infarction; PCI, percutaneous coronary intervention; CABG, coronary artery bypass grafting; HDL, high-density lipoprotein; PRA, plasma renin activity; LVMI, left ventricular mass index; FS, fractional shortening; CAS, carotid artery stenosis; NASCET, North American Symptomatic Carotid Endarterectomy Trial; n.s., not significant.

Atherosclerosis is considered a general process, leading to both diffuse subclinical changes in the whole vasculature and localized lesions causing clinical syndromes. This view is strengthened by observations from population studies, where atherosclerotic lesions in the common carotid artery, as visualized by B-mode ultrasound, appear to represent the total atherosclerotic burden. Several studies have revealed that the extent of extracranial carotid intimal thickening is correlated with coronary (*8*) and femoral artery (*9*) disease but not with renal artery diseases.

This report describes the prevalence and severity of renal artery disease in patients with severe carotid artery atherosclerosis. The features of atherosclerotic involvement in these arterial segments were analyzed to address three questions: 1) Is there an association between carotid atherosclerotic disease and renal arteries? 2) What clinical factors correlate with the severity of renal artery atherosclerosis in patients with carotid artery disease? 3) What are the appropriate guidelines for screening renal artery disease in patients presenting with carotid artery stenosis (CAS)?

Methods

Patients

We conducted a prospective analysis to evaluate renal function and renal artery disease in patients with severe CAS at the National Cardiovascular Center, Suita, Japan. Between February and August 2006, 41 consecutive patients (32 men and 9 women), whose confirmed CAS had been confirmed by duplex scanning and cerebral angiography and who had been admitted to the Department of Neurosurgery for a preoperative examination, were prospectively included. Tables 1 and 2 summarize the patients' baseline clinical features and renal characteristics. All patients gave informed consent to participate in this study.

Hypertension was defined as a patient's use of antihypertensive medication and/or a well-established history of elevated casual blood pressure ≥140/90 mmHg. Diabetes mellitus was defined as a patient's use of oral hypoglycemic

Table 2. Renal Function of the Study Subjects

Variables	Patients with RAS (n=11)	Patients without RAS (n=30)	p value
BUN (mg/dL)	23±11	16±5	<0.05
Creatinine (mg/dL)	1.5±0.5	0.9±0.3	<0.01
Uric acid (mg/dL)	6.7±1.4	5.6±1.4	<0.05
Creatinine clearance (mL/min)	55±19	70±23	<0.05
CrCl<60 mL/min (n (%))	7 (60)	10 (33)	<0.05
Urinary albumin excretion (mg/g Cr)	26.0±45.9	72.5±150.2	n.s.
Renal Doppler sonography			
Renal length (no-stenosis) (mm)	119±8	103±13	n.s.
Renal length (stenosis) (mm)	99±10*	—	
Peak velocity (no-stenosis) (m/s)	1.1±0.4	0.8±0.2	<0.05
Peak velocity (stenosis) (m/s)	2.8±1.1**,##	—	
Mean RI (no-stenosis)	0.77±0.10	0.67±0.07	<0.05
Mean RI (stenosis)	0.77±0.08#	—	
Degree of RAS (no-stenosis) (%)	18±27	—	
Degree of RAS (stenosis) (%)	78±13**	—	

RAS, renal artery stenosis; BUN, blood urea nitrogen; CrCl, creatinine clearance; Cr, creatinine; RI, resistance index. *p<0.05 *vs*. no-stenosis, **p<0.005 *vs*. no-stenosis, #p<0.05 *vs*. patients without RAS, ##p<0.01 *vs*. patients without RAS. n.s., not significant.

agents or insulin, and/or having a fasting plasma glucose level >126 mg/dL. Hyperlipidemia was defined as having a serum cholesterol level >220 mg/dL or taking lipid-lowering agents. Coronary artery disease was diagnosed by coronary angiography or stress scintigraphy. Myocardial infarction was diagnosed by QRS change on ECG (abnormal Q or poor R progression), hypokinesis of wall motion on echocardiogram, or elevation of serum myocardial enzyme (creatinine kinase-MB) levels. Cerebral vascular disease (cerebral infarction or hemorrhage) was diagnosed according to clinical history, neurological examination, and findings of computerized tomography. Peripheral vascular disease was diagnosed by exertional claudication and/or any of the following: resting pain, prior revascularization procedure to legs, absent or diminished pulse in legs, ankle/brachial pressure index less than 0.9. Current tobacco use was defined as use within 2 weeks prior to participation in the study.

Clinical data and laboratory data that included body mass index (BMI), arterial blood pressure, urinary albumin excretion, blood urea nitrogen, serum creatinine, uric acid, total cholesterol, high-density lipoprotein (HDL)-cholesterol, triglycerides, and fasting plasma glucose were measured.

The carotid arteries were ultrasonographically assessed using an ATL Ultramark 9 HDI unit (Advanced Technology Laboratories, Bothell, USA) with a linear-array pulsed-wave transducer operating at 5.0 to 10.0 MHz. Imaging was performed while the subject lay in a supine position with the head turned away from the side being scanned and the neck extended. The right and left common carotid arteries, the carotid bifurcation-bulb area, and both internal carotid arteries were scanned. Using power Doppler imaging, we measured the degree of diameter stenosis in longitudinal views and the percentage of area reduction (area stenosis) (*8*, *10*).

Selective cerebral angiography was performed by using the intracranial digital subtraction technique *via* the femoral artery route with selective catheterization of the extracranial arteries. The linear-based method of the North American Symptomatic Carotid Endarterectomy Trial (NASCET) was used to measure reduction of the internal carotid artery diameter (*11*).

Ultrasound examinations of renal arteries using a duplex Doppler apparatus (SSA-380A, Toshiba Inc., Tokyo, Japan; and System 5, Aloka, Tokyo, Japan) were performed with the subject in a supine position in the morning after overnight fasting. The apparatus obtained images with a 2.5–3.75 MHz convex or sector array probe in both real-time color-coded Doppler and pulsed Doppler modes. The abdominal aorta was evaluated initially. The peak systolic flow velocity (PSV) and the end-diastolic flow velocity (EDV) of the renal arteries and segmental arteries were evaluated. The resistance index (RI) (=(PSV−EDV)/PSV) was calculated as the average of six total measurements obtained from selected segmental arteries from the upper, middle, and lower parts of the bilateral kidneys (*12*). The severity of stenosis in each renal artery was classified according to previously validated criteria based on the PSV in the renal artery and the renal-aortic ratio (RAR), defined as the ratio of the PSV in the renal artery at the site of stenosis to the PSV in the adjacent abdominal aorta (*13*). To estimate the severity of stenosis in a renal artery, these criteria classify renal artery narrowing into four categories: 1, normal (0% diameter stenosis): PSV<1.8 m/s and RAR<3.5; 2, less than 60% diameter stenosis: PSV>1.8 m/s and RAR<3.5; 3, 60% diameter stenosis or greater: PSV>1.8 m/s and RAR>3.5; and 4, occlusion (*13*). Cranio-caudal measure-

ment of each kidney was performed. The intra-assay and inter-assay coefficients of variation of these renal Doppler parameters were 2.7% and 3.2% in RI, respectively (*12*).

MRI or abdominal angiography was performed to determine the degree of maximal RAS in patients with renal artery narrowing grade 3 or 4 (*14*).

Creatinine clearance (CrCl) was estimated with the Cockcroft-Gault formula, as follows (*15*):

$$\text{Estimated CrCl (mL/min)} = (140 - \text{age})(\text{weight in kg})/72 \times \text{serum creatinine (mg/dL)}.$$

This formula was adapted for women by multiplying by 0.85.

Urine without any sign of urinary infection was collected at 24-h intervals, and urine albumin excretion (UAE) was measured by radioimmunoassay.

M-mode echocardiograms with the patient in the recumbent position were performed to estimate the left ventricular mass index (LVMI, g/m^2) and fractional shortening (FS, %).

Statistical Analysis

All analyses were performed with the program StatView 5.0 (SAS Institute, Cary, USA). Differences in baseline characteristics between the groups were compared by ANOVA for continuous variables and by the χ^2 test for categorical variables. A value of $p<0.05$ was considered statistically significant. Risk factors for the presence of RAS were evaluated using logistic regression analysis. The results are expressed as means±SD.

Results

The patients' baseline characteristics are listed in Table 1. Patients with severe CAS were classified into two groups based on the results of duplex scanning of the renal arteries. Patients with grade 1or 2 were classified as not having RAS ($n=30$). Patients with grade 3 or 4 were classified as having RAS ($n=11$), and MRI or abdominal angiography was performed in the RAS group to clarify the degree of maximal RAS. A total of 27% of CAS patients were considered to have RAS. Age, gender, BMI, hypertension, diabetes, and hyperlipidemia did not differ significantly between the groups. Cardiovascular diseases such as coronary artery disease ($p<0.005$) and peripheral vascular disease ($p<0.05$) were commonly present in the RAS patients, and this group also included many current smokers ($p<0.05$). Systolic blood pressure, diastolic blood pressure, and pulse pressure did not differ between the groups. Cardiac function and the degree of CAS were not significantly different.

The patients' renal function characteristics are listed in Table 2. Median serum creatinine concentrations were 1.5±0.5 mg/dL in the patients with RAS and 0.9±0.3 mg/dL in the patients without RAS ($p<0.01$). Blood urea nitrogen and uric acid were significantly higher ($p<0.05$), and estimated CrCl was significantly lower, in the RAS patients ($p<0.05$). A total of 64% of RAS patients had estimated CrCl less than 60 mL/min (the same as chronic kidney disease grade 3). On the other hand, urinary albumin excretion did not differ significantly.

The renal Doppler sonography showed that the renal length of kidneys without stenosis was not different between the RAS and non-RAS groups. In the RAS group, renal length and peak velocity differed significantly between the kidneys with stenosis and those without. Peak velocity and RI of kidneys without stenosis differed between the groups. In the RAS group, the RI did not differ between the kidneys with stenosis and those without. Angiography showed that the degrees of RAS in the RAS group were 18% in the kidneys without stenosis and 78% in those with stenosis. These findings showed that, in the RAS group, kidneys with stenosis had severe RAS, atrophic kidney, and renal dysfunction; contralateral kidneys had moderate RAS and renal dysfunction but did not have atrophic kidney.

Logistic regression analysis was performed to clarify the univariate risk factors for the presence of RAS (Table 3). Blood urea nitrogen (BUN), creatinine, and the presence of coronary artery disease or peripheral vascular disease were evaluated as risk factors. Risk factors including estimated CrCl, CrCl less than 60 mL/min, RI more than 0.8, uric acid, and current smoking tended to be significant.

Multivariate risk factors were determined using logistic regression analysis. Current smoking, estimated CrCl less than 60 mL/min, and the presence of coronary artery disease were identified as risk factors (Table 4).

Discussion

We evaluated the prevalence and severity of RAS in patients with severe CAS and found the following. 1) There was a clear association between the carotid and renal arteries in atherosclerotic disease. More than one quarter of the patients with significant CAS had RAS. 2) RAS patients had impaired renal function compared with patients without RAS. Cardiovascular diseases and current smoking were frequent in the RAS patients. 3) The risk factors for the presence of RAS were current smoking, renal dysfunction, and coronary artery disease.

RAS has been recognized as a cause of secondary hypertension and chronic renal failure. Our previous study indicated that RAS is relatively common in patients with myocardial infarction, stroke, and abdominal aortic aneurysm (*5–7*). We also revealed that hypertension, proteinuria, and renal dysfunction, but not older age, were related to the presence of RAS (*5–7*). A recent study indicated that older age and renal impairment were independent predictors of atherosclerotic RAS in Japanese patients with atherosclerotic risk factors (>40 years of age plus hypertension, dyslipidemia, or diabetes mellitus) (*16*). In patients with apparent cardiovascular diseases such as myocardial infarction, stroke, or aortic aneu-

Table 3. Univariate Risk Factors for the Presence of RAS Based on Logistic Regression Analysis

Variables	RR	95% CI	*p* value
Age (years old)	1.06	0.96–1.16	n.s.
Male gender (yes)	3.64	0.4–33.1	n.s.
Systolic blood pressure (mmHg)	1.04	0.97–1.10	n.s.
Diastolic blood pressure (mmHg)	0.99	0.92–1.06	n.s.
Urinary albumin excretion (mg/g Cr)	0.99	0.98–1.01	n.s.
BUN (mg/dL)	1.13	1.01–1.25	<0.05
Creatinine (mg/dL)	18.1	1.7–192.2	<0.05
Uric acid (mg/dL)	0.71	0.98–2.99	0.06
CrCl (mL/min)	0.97	0.93–1.01	0.07
CrCl<60 mL/min (yes)	3.50	0.83–14.84	0.09
PRA (ng/mL/h)	1.00	0.77–1.29	n.s.
Hypertension (yes)	2.0	0.21–19.3	n.s.
Diabetes (yes)	0.98	0.24–3.93	n.s.
Hyperlipemia (yes)	1.63	0.63–7.48	n.s.
Coronary artery disease (yes)	10.0	1.8–56.0	<0.01
Cerebral vascular disease (yes)	1.61	0.24–4.26	n.s.
Peripheral vascular disease (yes)	7.2	1.34–38.9	<0.05
Current smoking (yes)	8.13	0.92–72.0	0.06
Number of CVD	2.05	1.02–4.13	<0.05
RI>0.8 (yes)	5.50	0.90–31.60	0.06

RAS, renal artery stenosis; RR, relative risk; CI, confidence interval; Cr, creatinine; BUN, blood urea nitrogen; CrCl, creatinine clearance; CVD, cardiovascular disease; RI, resistance index; n.s., not significant.

Table 4. Multivariate Risk Factors for the Presence of RAS Based on Logistic Regression Analysis

Variables	RR	95% CI	*p* value
Current smoking (yes)	18.7	1.24–281.3	<0.05
CrCl<60 mL/min (yes)	9.4	1.17–74.8	<0.05
Coronary artery disease (yes)	7.0	1.00–48.9	<0.05

RAS, renal artery stenosis; RR, relative risk; CI, confidence interval.

rysm, older age was a common finding independent of the presence of RAS, and therefore older age did not become a risk factor for RAS. In this study, 11 patients with significant CAS had RAS. Renal dysfunction, current smoking, and cardiovascular diseases were frequently apparent in the patients with RAS, whereas older age was not a risk factor.

On the other hand, renal dysfunction has been considered a risk factor in cardiovascular operations and for survival in cardiovascular diseases (*17–20*). In the treatment of cardiovascular diseases, the evaluation of renal condition is becoming more important and necessary.

CAS has been studied mainly in the field of neurosurgery, and these studies have focused on methods of repair, patient survival, and risk factors. In 1991, NASCET proved conclusively the effectiveness of carotid endarterectomy in patients with symptomatic internal CAS which was greater than 70% (*11*). In patients with RAS, several studies (*21–23*) showed the relationship between renal and carotid arteries. However, there are few studies about renal artery disease in patients with CAS. The occurrence of renal cholesterol embolism after cerebrovascular procedure was reported previously (*24*). We also reported previously that renal function sometimes deteriorated in patients with abdominal aortic aneurysm after an elective operation, although the development of end-stage renal failure is rare (*7*). RAS is considered to carry at least some responsibility for postoperative renal dysfunction (*7*). During abdominal aortic aneurysm surgery, systemic blood pressure may fall due to systemic anesthesia or blood loss, and renal blood flow may be reduced due to cross cramping of the supra-renal abdominal aorta. Compared with cases of abdominal aortic aneurysm, systemic blood pressure may be stable in carotid endarterectomy cases. However, after carotid endarterectomy, the blood supply to the brain is increased due to the improvement in CAS, and then hyperperfusion of cerebral blood flow occurs, even with normal blood pressure. In this condition, the reduction of systemic blood pressure using antihypertensive drugs is considered necessary to improve the hyperperfusion of cerebral blood flow while also inducing a reduction in renal perfusion pressure; renal dysfunction or acute renal failure may occur in cases with RAS. A longer duration of renal ischemia may cause a transient increase in creatinine or a reduction in urine output. Therefore, it is considered useful to obtain preoperative information about renal artery disease in patients with severe CAS undergoing carotid endarterectomy.

In this study, we showed that patients with significant CAS had RAS with almost the same frequency as ischemic heart disease. The risk factors for RAS were the presence of ischemic heart disease, smoking, and renal dysfunction, and these are considered useful for recognizing the presence of RAS. In cases with bilateral RAS, the RAS revascularization procedure may be useful and necessary to improve renal deterioration after carotid endarterectomy. In cases with severe renal parenchymal damage, neither overreaching blood pressure reduction nor antihypertensive medication to treat the renin-angiotensin system is suitable. In these complicated cases, it is necessary to consult a nephrologist to assess the patient's renal function.

Both RAS and CAS occurred as a result of systemic atherosclerosis. Using ultrasonography, it is considered easier to evaluate CAS than RAS. The RAS patients were often asymptomatic, and the diagnosis of RAS needed a more complicated examination, such as Doppler sonography, MRI, or CT. It is important to consider the presence of RAS in patients with severe CAS, as this will be useful for preventing hypertension and renal failure.

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