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ORIGINAL ARTICLE

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More coronary artery stenosis, more cerebral artery stenosis? A simultaneous angiographic study discloses their strong correlation

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Abstract Cerebral artery stenosis (CAS) has the same pathogenesis as coronary artery disease (CAD), but the coexistence of these two diseases has been rarely reported. To detect coexistent CAS in CAD patients, we conducted a study of simultaneous coronary and cerebral angiography. Of the 663 consecutive newly diagnosed CAD patients who had not yet been explored to have CAS, 80 were admitted to undergo angiography of bilateral carotid and vertebral system during the same procedure. We defined significant vascular stenosis, either located intracranially or extracranially, as the lesions of diameter stenosis more than 50%. Association between carotid or vertebral stenosis and their potential risk factors were also analyzed. Of our patients, 18 (22.5%) had significant extracranial vascular stenosis, 14 (17.5%) suffered from intracranial stenosis, and 20 (25%) had both. Only 28 patients (35%) had no significant intracranial or extracranial stenosis. None of the demographic parameters as hypertension or diabetes showed significant differences between the cerebral patent group and the CAS group, except for the number of coronary stenotic vessels (1.71 ± 0.81 versus 2.69 ± 0.64, $P < 0.001$). The number of coronary stenotic vessels is correlated well to the number of cerebral stenotic lesions ($r = 0.562$, $P < 0.001$). Besides, 8 of the cerebral stenotic patients and 2 of the cerebral patent patients had ischemic stroke previously. We conclude the CAS is coexistent in more than half of the CAD patients in this study. Our study also implies a proportional increase in the severity of CAS to CAD severity.

Key words Coronary artery disease · Angiography · Cerebral artery stenosis

Introduction

Cerebral artery stenosis, either extracranially or intracranially, shared the common pathogenesis with coronary artery disease. There have been several articles reporting the coexistence rate of these two diseases, but most of them were based on non-invasive imaging methods as carotid duplex study,[1–6] which are relatively less accurate than the gold standard, digital subtraction angiography (DSA).[7] Although the magnetic resonance angiography (MRA) is believed to be a good alternative to DSA in providing intracranial vasculature information noninvasively, its sensitivity and specificity in this field has remained insufficient in recent head-to-head comparative reports with DSA.[8,9] Therefore, to define the accurate coexistent rate of coronary artery disease (CAD) and cerebral artery stenosis (CAS), we planned to conduct a simultaneous cerebral angiography for patients with coronary artery disease.

Patients and methods

From November 16, 2004 to April 14, 2005, 633 patients were diagnosed to have coronary artery disease by coronary angiography (CAG) in our hospital with their cerebral vasculature not yet studied. Before the CAGs were performed, all of the patients who were going to undergo the procedure would be asked permission to have a simultaneous cerebral angiography during the same procedure. Only 93 of those patients with CAD admitted to have a further cerebral angiography after procedure-related risk abstained. However, in order to avoid unnecessary compli-

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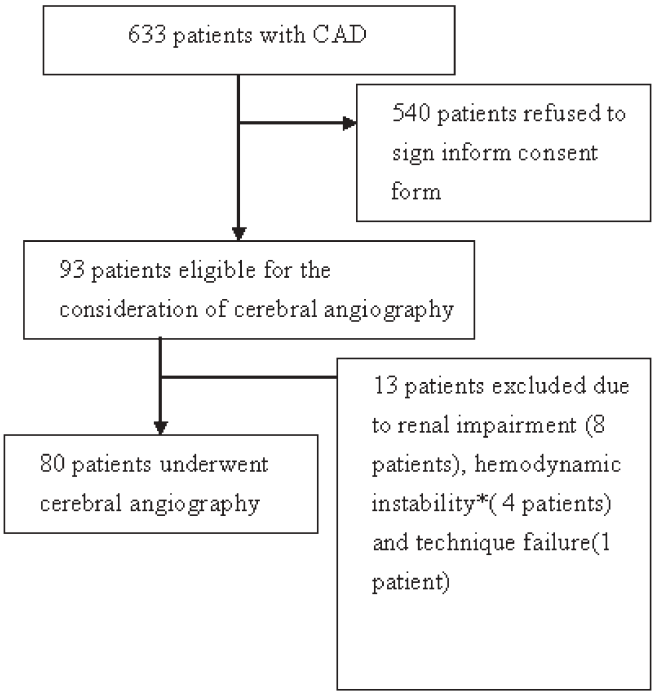

Fig. 1. Process of the patients enrollment. *Cerebral angiography could not be conducted extemporaneously in those patients with hemodynamic instability

Table 1. Demographic parameters of the sample (80 patients) and the remaining patients

	Sample (n = 80)	Remaining patients (n = 553)	P value
Age (years)	66.7 ± 11.7	67.5 ± 15.4	0.93
Sex (Female: male)	27:53	180:373	0.79
Diabetes	31	206	0.41
Hypertension	64	377	0.06
Cholesterol (mg/dl)	191.7 ± 43.1	191.1 ± 50.3	0.918
Triglyceride (mg/dl)	182.2 ± 126.8	166.9 ± 103.3	0.253

cations, we excluded 13 more patients due to other medical reasons such as recent stroke, intracranial atherosclerosis diagnosed already by other methods, etc. The whole process of patients' enrollment is demonstrated in Fig. 1. Our institutional review committee approved the study protocol.

All the baseline demographic data of these patients were recorded including age, previous stroke history, hypertension (previously diagnosed and treated or systolic pressure >140 mmHg and/or diastolic pressure >90 mmHg), diabetes (previously diagnosed and treated or fasting glucose >7 mmol/l), serum levels of cholesterol and triglyceride, history of previous coronary artery bypass surgery (CABG), and smoking status (current smoker or abstinence within 1 year). To detect any selection bias in our sampling process, we analyzed the basic demographic data of the 80 participants selected and the remaining 583 patients as in Table 1.

All patients underwent coronary angiography of right coronary artery and left coronary artery. Significant coronary artery stenosis is defined as diameter stenosis more than 50% according to the criteria of Coronary Artery Bypass Graft Surgery Trialists Collaboration.[10] We define the extent of coronary artery disease as the number of significant stenotic coronary arteries (i.e., 1 means single-vessel disease, 2 means 2-vessel disease and purely left main disease, and 4 means left main and triple-vessel disease).

Digital subtraction cerebral angiography (i.e., both carotid and vertebrobasilar system) was performed for all patients enrolled with a Siemens BICOR PLUS angiography system or Philips Integris BH 5000 system. For every stenotic lesion either extracranially or intracranially, qualification analysis software (Siemens, Quancor, QCA [CASS II, v4.0], or Philips manual analysis online) was used for lesion measurement. According to the criteria of Warfarin-Aspirin Symptomatic Intracranial Disease Study for Stroke,[11] we defined significant stenosis as diameter stenosis more than 50%–99% of any major intracranial artery (carotid artery, MCA stem (M1), vertebral artery, and basilar artery) by angiography. The stenosis of the cervical portion of internal carotid artery and the extracranial vertebral arteries were defined according the same criteria.

Furthermore, the patients were classified into the cerebral patent group (no significant stenosis of vertebrobasilar or carotid system) and the cerebral stenotic group (at least one significant stenosis in carotid or vertebral system). We defined cerebral stenotic status as the number of all the lesions with significant stenosis.

Univariate analyses was conducted for each of the demographic factors as well as coronary stenotic status (1, 2, 3, or 4) between cerebral stenotic patients and the cerebral patent group with the Chi-square test (for the nominal variables such as sex ratio, smoking status, diabetes mellitus, stroke history, and hyperlipidemia) or independent t-test (for the rational variables such as fasting sugar and lipid level) except for CAD status, which was analyzed with the nonparametric Mann–Whitney U method. On the other hand, the correlation coefficient between cerebral stenotic status and each of the demographic parameters was computed. The Kruskal–Wallis nonparametric method was used for depicting the box plots of CAD status and cerebral stenotic status. All procedures were conducted with SPSS 11th edition computerized software, and a P value less than 0.05 was considered significant.

Results

There was no significant difference in major demographic parameters (gender, diabetes, hypertension, and lipid profile) between the sample group and the remaining patients (Table 1).

In all 80 patients with CAD, 18 (22.5%) had extracranial atherosclerotic stenosis, 14 (17.5%) suffered from intracranial atherosclerotic stenosis, and 20 (25%) had both. In total 65% of our CAD patients had significant cerebral stenosis, either intracranially or extracranially. Only 28 of

Table 2. Demographic data of cerebral patent group and cerebral stenotic group

	Cerebral patent group (n = 28)	Cerebral stenotic group (n = 52)	P value
Age (years)	63.07 ± 10.22	67.52 ± 12.23	0.105[b]
Sex (M:F)	20:8	34:18	0.582[a]
Diabetes	12 (46.2%)	24 (42.9%)	0.777[a]
Hypertension	23 (82.1%)	42 (80.8%)	0.828[a]
Smoking	9 (17.3%)	8 (28.6%)	0.353[a]
Family history	8 (22.9%)	10 (22.2%)	0.946[a]
Fasting sugar (mg/dl)	155.33 ± 80.82	175.31 ± 103.60	0.386[b]
Cholesterol (mg/dl)	189.00 ± 37.74	197.72 ± 60.83	0.501[b]
Triglyceride (mg/dl)	206.67 ± 158.33	176.46 ± 120.65	0.352[b]
Extent of coronary artery disease	1.71 ± 0.81	2.69 ± 0.64	<0.001[c]
CABG history	3 (10.7%)	11 (21.2%)	0.241[a]
Stroke history	2 (7.1%)	8 (15.4%)	0.288[a]
Major infarct	1	6	
TIA	1	2	

CABG, coronary artery bypass grafting; TIA, transient ischemic accident
[a] Calculated with Pearson's Chi-square method
[b] Calculated with independent t-test
[c] Calculated with non-parametric Mann–Whitney method

Table 3. Correlation of cerebral stenosis status and several demographic factors

	Correlation coefficient[a]	P value[b]
Age	0.259	0.02
Fasting sugar (mg/dl)	0.152	0.182
Cholesterol (mg/dl)	0.002	0.965
Triglyceride (mg/dl)	−0.121	0.295
CAD status	0.562	<0.001

[a] Pearson product–moment correlation coefficient
[b] Pearson correlation coefficient significance (2-tailed)

them (35%) had completely patent cerebral angiography. After univariate analysis of all the demographic factors and CAD status between diseased and patent cerebral patients, we found only the coronary stenotic status (i.e., number of stenotic vessels) to be significantly different between the two groups (Table 2). On the other hand, 8 of the cerebral stenotic patients and 2 of the cerebral patent patients had a history of previous ischemic stroke.

Similarly, the correlation analysis revealed only the coronary stenotic status and age to correlate well with cerebral stenotic status (Table 3). That is to say, the greater the number of stenotic coronary vessels, the more was the number of stenotic cerebral lesions, either intracranially or extracranially (Fig. 2). If we view the relation of coronary status to the number of extracranial cerebral stenoses (Fig. 3), and the number of intracranial cerebral stenoses separately (Fig. 4), we can still see the same trend. Moreover, the age of the patients showed a minor association with coronary stenotic status.

Of all the 95 cerebral stenotic lesions, 41 (43.15%) were located extracranially and 54 (56.85%) intracranially (Table 4). On the other hand, 33 (34.7%) lesions were located in vertebrobasilar system and 62 (65.3%) in the carotid system.

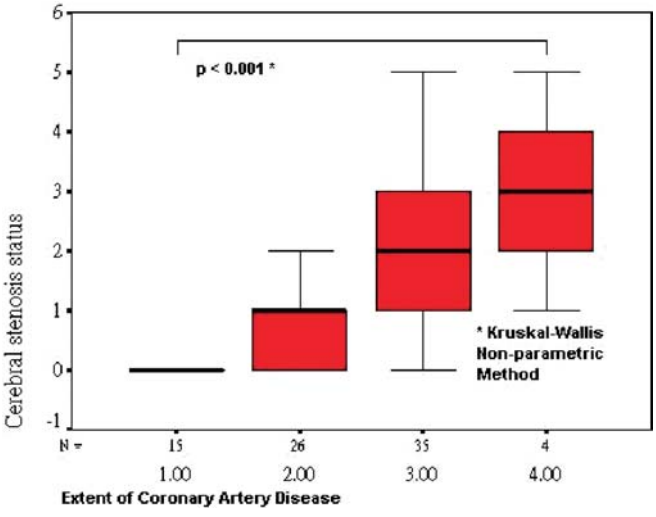

Fig. 2. Cerebral stenotic status in different coronary stenotic status (box-and-whisker plots). Cerebral stenosis status means the number of all the cerebrovascular lesions with significant stenosis, and extent of coronary artery disease means the number of significant stenotic coronary arteries (i.e., 1 stands for single-vessel disease and 4 for left main and triple-vessel disease)

Discussion

Coexistence of atherosclerosis in different vascular systems has been reported for decades. Of the patients with peripheral artery occlusive disease, 24%–71% have CAD as well,[12–14] and about 27%–48% suffer from coexistent carotid stenosis.[12–15] On the other hand, nearly one tenth of CAD patients are associated with peripheral artery occlusive disease.[16] However, CAD and cerebral artery stenosis not only have been noted to be coexisting with each other, but also have strongly prognostic significance.[1–6]

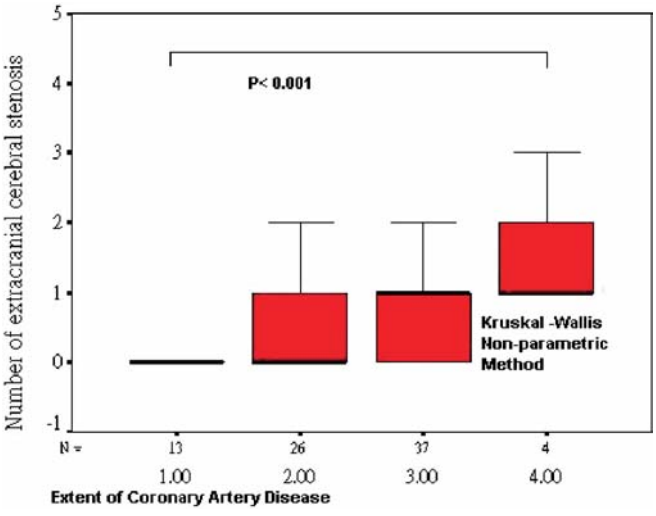

Fig. 3. Number of extracranial cerebral stenosis in different "extent of coronary artery disease" (box-and-whisker plots). Number of extracranial cerebral stenosis means the number of all the extracranial cerebrovascular lesions with significant stenosis, and extent of coronary artery disease means the number of significant stenotic coronary arteries (i.e., 1 stands for single-vessel disease and 4 for left main and triple-vessel disease)

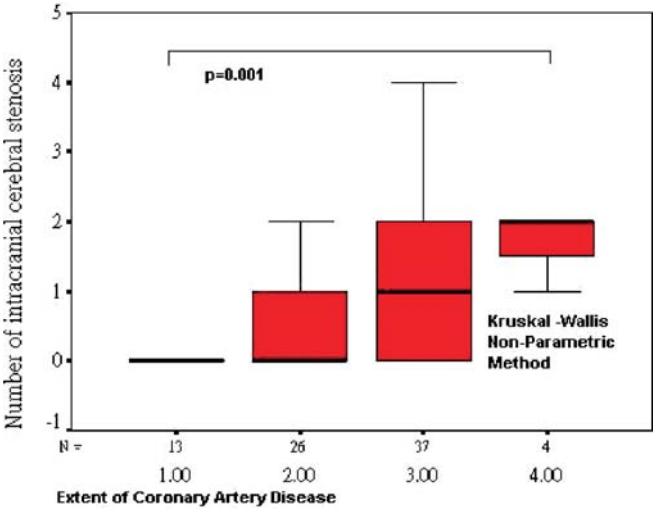

Fig. 4. Number of intracranial cerebral stenosis in different "extent of coronary artery disease" (box-and-whisker plots). Number of intracranial cerebral stenosis means the number of all the intracranial cerebrovascular lesions with significant stenosis, and extent of coronary artery disease means the number of significant stenotic coronary arteries (i.e., 1 stands for single-vessel disease and 4 for left main and triple-vessel disease)

First of all, patients undergoing carotid endaterectomy often have cardiac events afterward or prove to have silent CAD.[1–3] An angiography study of patients with extracranial cerebrovascular disease showed that 37% of them suffered from CAD.[4] Even the noninvasive ultrasound parameters of carotid artery, such as intima-media thickness or the B-mode score of arterial wall, can predict the risk of coexistent CAD.[5,6]

On the other hand, it has been reported that about 13%–27.6% of CAD patients have carotid artery stenosis,[17–19] and the prevalence rate of proximal vertebral lesions in CAD patients is up to 41.6%.[20] Such a high coexistent rate also imposes a significant prognostic impact on these patients. For those patients undergoing coronary artery bypass grafting surgery (CABG), the post-procedure stroke rate is at least 1%–5.2%.[21–23] If the minor neurological adverse outcome is taken into account, its prevalence can be much higher.[24] Consequently, Suematsu et al. proposed surgical revascularization of the carotid system before CABG in patients with coexistent CAD and carotid occlusion to prevent neurological events.[25]

Table 4. Locations of cerebral stenotic lesions

Location of lesions	No. of lesions	Percentage
Vertebral orifice	19	20.0
Common carotid	2	2.1
Cervical ICA	20	21.1
Intracranial vertebral artery	6	6.3
Basilar artery	8	8.4
Petrous ICA	14	14.7
Cavernous ICA	11	11.5
Anterior cerebral artery	5	5.3
Proximal middle cerebral artery	10	10.5
Total	95	100

ICA, internal carotid artery

Compared with standard cerebral angiography, noninvasive scanning methods used by most of the studies listed above, such as transcranial Doppler, can provide only moderate accuracy in this field.[7] As a matter of fact, there have been two magnetic resonance angiography (MRA)-based studies published about patients scheduled for CABG. Uehara et al. reported significant prevalence of intracranial artery stenosis and cervical carotid stenosis (21.2% and 16.6%, respectively) in 151 consecutive patients who were scheduled for CABG.[26] Yoon et al. reported a much higher prevalence rate, i.e., 109 of their 201 consecutive patients (54.2%) having intracranial and/or extracranial cerebral artery stenosis, which was strongly associated with postoperative neurological complications.[27] Interestingly, the same group has just reported their findings in 2006 that the correlation of coronary artery stenosis with extracranial carotid atherosclerosis is stronger than that of coronary artery stenosis with intracranial carotid atherosclerosis.[28] This differs from our findings, and we believe the discrepancy of these two studies results from the diagnostic tools of the carotid atherosclerosis: the Yoon et al. study uses the transcranial Doppler test as the diagnostic standard and it is relatively less accurate than DSA.

Compared with DSA, which is the gold standard for the diagnosis of intracranial atherosclerosis, the sensitivity and specificity of MRA might not be good enough. A head-to-head comparative study of MRA and DSA, which evaluated intracranial vessels in acute stroke and transient ischemic attack (TIA) patients, showed the sensitivity and specificity of MRA to be around 70%.[8] Moreover, another recent report showed the positive predictive rate of MRA to be even less than 70%.[9] Having conducted simultaneous coronary and cerebral angiography, our study was able to

define more accurately the prevalence of CAS in advanced CAD patients than all previous studies. From our findings, it is reasonable to suspect one patient of multiple-vessel coronary disease to have as high as a 50% risk of coexistent cerebral artery stenosis, which might lead to neurological complications in the future.

Another important finding of our study is the strong correlation between the coronary stenosis status and the number of cerebrovascular lesions. Since both of them share the same atherosclerotic process, this finding really makes sense. Some indirect evidence in favor of this point has been published already. Lazar et al. reported the presence of more than two coronary arteries with more than 50% narrowing as being an independent predictor of neurological events after percutaneous coronary intervention.[29] Furthermore, Geroulakos et al. noted that there is even a significantly linear trend between carotid intima-media thickness and the number of stenotic coronary vessels.[30]

Ethnic discrepancy has been demonstrated in the prevalence of intracranial stenosis in the Northern Manhattan Stroke Study,[31] which showed that the non-white population seems to have 5 times higher risk of suffering from intracranial stenosis. Whether there is any race/ethnic reason underlining the findings of our study is debatable. However, Su and his colleagues demonstrated in a community-based duplex study that the prevalence of carotid stenosis (defined as diameter stenosis more than 50%) in Taiwan is only around 3.7%, which is much lower than the CAD patients enrolled in the present study.[32] Actually, the prevalence of cerebral stenosis of our CAD patients, either intracranially or extracranially, is close to the level of another MRA study of Taiwanese ischemic stroke patients, which showed the prevalence both of ECAD and ICAD of around 42%.[33]

Coexistent cerebrovascular disease imposes prognostic significance on patients with CAD.[34] We believe further follow-up of our patients will disclose the same trend, despite it taking time to achieve. Furthermore, approximately 85% of our cerebral stenotic patients still denied any history of stroke and remain asymptomatic neurologically. In other words, they are the victims of asymptomatic carotid stenosis and/or asymptomatic intracranial atherosclerotic stenosis. The Wafarin-Aspirin Symptomatic Intracranial disease trial has demonstrated the natural history of symptomatic intracranial atherosclerosis very well,[35] but there is no study on asymptomatic intracranial disease. As a result, our study has enlightened several directions in the future, i.e., natural history of asymptomatic ICAD and ECAD, and possible primary prevention in these groups. However, we need to enroll more patients for these purposes.

Finally, another issue to be discussed is the safety of the cerebral angiography routinely performed by cardiologists for CAD patients. Fayed et al. have demonstrated the safety of cerebral angiography performed by experienced cardiologists.[36] After comparison with previous reports, there is no significant difference of procedure-related complication rate between the results of Fayed et al. and the experience of interventional radiologists.[37]

Conclusions

More than half of the CAD patients in this study had coexistent CAS. Our data also imply a proportional increase of the severity of CAS to CAD severity. Moreover, such cerebral stenotic patients are strongly associated with a history of previous ischemic stroke.

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