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# Evaluation of crossed cerebellar diaschisis in 30 patients with major cerebral artery occlusion by means of quantitative I-123 IMP SPECT

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Quantitative crossed cerebellar diaschisis (CCD) and the correlation with a reduction in supratentorial regional cerebral blood flow (rCBF) and cerebrovascular reserve capacity (CVR) were investigated in clinically stable patients with major cerebral artery occlusion by the iodine-123-*N*-isopropyl-*p*-iodoamphetamine (I-123 IMP) single photon emission computed tomography (SPECT) method. Thirty patients with major cerebral artery occlusion underwent SPECT by the I-123 IMP autoradiographic method. Regional CBF was measured in the cerebral hemisphere, frontal and parietal lobes, temporo-parietal lobe, and cerebellum both at rest and after administration of acetazolamide. Eighteen of 30 patients (60%) had CCD. CCD was significantly related to magnetic resonance imaging evidence of infarction. Quantitative CCD was 17% and the CVR in the cerebellum was preserved in patients with CCD. There was a significant difference in CBF and CVR between the affected and normal sides in all regions of interest in the patients without CCD [CBF (ml/100 g/min): hemisphere (H), normal side (N): $31.4 \pm 6.8$, affected side (A): $27.5 \pm 7.4$; $p < 0.05$. CVR: H, N: $0.56 \pm 0.38$, A: $0.42 \pm 0.18$; $p < 0.01$]. CCD is common in patients with major cerebral artery occlusion, and quantitative I-123 IMP SPECT is helpful in detecting CCD in clinically stable patients with occlusion of major cerebral arteries.

**Key words:** $^{123}$I-IMP, crossed cerebellar diaschisis, cerebral artery, occlusion, SPECT

## INTRODUCTION

CROSSED CEREBELLAR DIASCHISIS (CCD) is a phenomenon of reversible matched decrease in blood flow and metabolism in the cerebellum of patients with contralateral supratentorial lesions and significant hemiparesis.[1–9] CCD is usually observed in patients with irreversible lesions, such as cerebral infarction, cerebral hematoma, and brain tumor,[10] but reversible lesions such as moyamoya disease and transient ischemic attack with cerebral artery occlusion are sometimes associated with CCD.[11–15] CCD has occurred during a temporary balloon occlusion test for predicting tolerance prior to permanent carotid sacrifice,[16–19] and transient CCD during barbiturate-induced unilateral cortical neuronal depression (Wada test).[20] Interruption of the corticopontocerebellar tract or other pathways between the cerebellum and supratentorial territory may be a causative factor.[1,2]

Most investigations of CCD have used qualitative or semi-quantitative methods, but two were based on positron emission tomography (PET) and single photon emission computed tomography (SPECT).[10–13] More quantitative investigations are necessary to evaluate the exact nature of CCD. The cost of examination by PET remains high, but several methods for quantification of regional cerebral blood flow (rCBF) with SPECT have been proposed.[21,22] The iodine-123-*N*-isopropyl-*p*-iodoamphetamine (I-123 IMP) method with one-point arterial blood

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sampling and acquisition of a single static SPECT scan has a close correlation with rCBF measured by PET with $H_2{}^{15}O$.[21]

This study investigated the occurrence, frequency and degree of CCD in clinically stable patients with major artery occlusion to observe whether CCD is correlated with the reduction in supratentorial rCBF measured by SPECT with the I-123 IMP method and to clarify the implications of CCD in major cerebral artery occlusive diseases.

## MATERIALS AND METHODS

*Patients*

Forty-two patients (30 in-patients and 12 out-patients) with suspected major cerebral artery occlusion were examined by cerebral angiography, magnetic resonance (MR) imaging, and I-123 IMP SPECT. Informed consent was obtained from the patients or relatives for all examinations. MR imaging was performed at the onset of symptoms and one month and three months after onset. Angiography was performed when the patient became stable, usually three weeks after onset. Thirty of the 42 patients, 24 men and 6 women aged 36 to 71 years (mean age 56.5 years), were found to have either unilateral middle cerebral artery (MCA) (18 patients) or internal carotid artery (ICA) (12 patients) occlusion. No patient had cerebellar signs, MR imaging evidence of infarction in the cerebellum, or angiographical steno-occlusive lesion of the vertebro-basilar artery. The initial symptoms were minor stroke in 9 patients, transient ischemic attack in 10 patients, and no neurological deficits, but chronic headache and dizziness were present in 9 patients. MR imaging showed no infarction in 10 patients, and lacunar infarction, small watershed infarction, or others in 20 patients.

*SPECT Study*

*Imaging procedures:* All patients underwent measurement of rCBF at rest and under acetazolamide stress by I-123 IMP autoradiography (ARG) SPECT.[22] Rest and acetazolamide stress SPECT were performed on separate days, usually within one week. Early and delayed SPECT images were obtained at 20 minutes and 3 hours, respectively, after injection of 111–222 MBq of I-123 IMP. Acetazolamide stress was induced by injection of 1000 mg acetazolamide 5–10 minutes before the administration of I-123 IMP. Distribution volume values were calculated by the table look-up method. A single point brachial arterial blood sample was collected from the side contralateral to the injection. All images were obtained with a Toshiba GCA 9300 A/DI triple head gamma camera (Toshiba, Tokyo) mounted with fan beam collimators (LESHR). The images were acquired as a 128 × 128 matrix by continuous rotation (10 rotations, 2 minutes/rotation) with 4 degree steps by using the triple energy window method. SPECT images were reconstructed by filtered back projection with Butterworth and Ramp filters (Butterworth and Ramp) after scattering correction. The thickness of the axial images was 6.8 mm. The mean interval from the onset of symptoms or initial examination to SPECT studies was 41.5 ± 21.3 days.

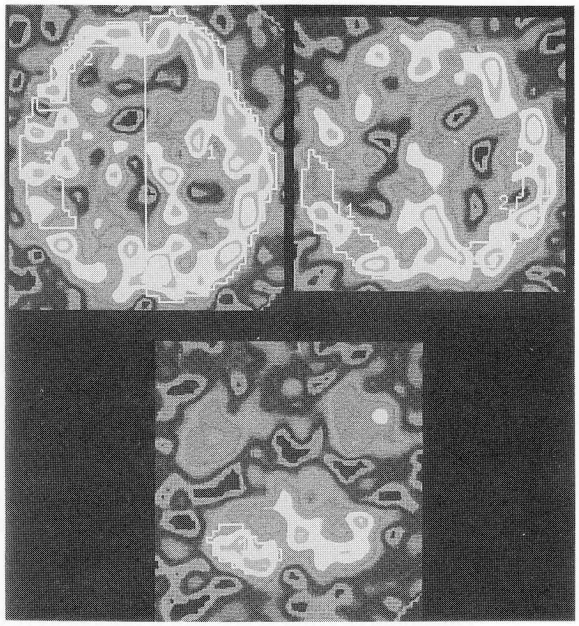

**Fig. 1** SPECT images illustrating the ROIs examined. Upper left: 1, hemisphere; 2, frontal lobe; 3, temporo-parietal lobe. Upper right: 1 and 2, parietal lobe. Lower row: 1, cerebellum.

*Image and data analysis:* The early and delayed images were compared side-by-side to identify the area of redistribution on the rest and acetazolamide stress images. Three axial images through the mid section of the cerebellum and the thalamus and basal ganglia just above the lateral ventricles were selected to create irregular regions of interest (ROIs) for the measurement of rCBF in the cerebellum, cerebral hemispheres, frontal lobes, parietal lobes, and the temporo-parietal lobe (Fig. 1). One investigator selected all these image sections and the same person also drew the ROIs to minimize the false registration of ROIs. The quantitative CCD was calculated from the following equation. CCD was considered to be present at values of more than 10%.[23–26]

$$\frac{\text{Cerebellar CBF (normal side)} - \text{Cerebellar CBF (affected side)}}{\text{Cerebellar CBF (normal side)}} \times 100\ (\%)$$

Cerebrovascular reserve capacity (CVR) was also measured with the following equation.

$$\frac{\text{rCBF (acetazolamide stress)} - \text{rCBF (at rest)}}{\text{rCBF (at rest)}}$$

Absolute rCBF and CVR in the normal and affected

**Table 1** Characteristics of patients

| | Crossed cerebellar diaschisis | |
|---|---|---|
| | (+) | (−) |
| Age* | 57.5 ± 11.3 | 55.0 ± 10.8 |
| Sex | | |
| Male | 12 | 10 |
| Female | 6 | 2 |
| Site of occlusion | | |
| ICA | 11 | 8 |
| MCA | 7 | 4 |
| Symptoms | | |
| Asymptomatic | 5 | 7 |
| TIA | 4 | 4 |
| Minor stroke | 9 | 1 |
| MR imaging | | |
| Normal | 3 | 7 |
| Lacunar infarction | 0 | 2 |
| Watershed infarction | 7 | 2 |
| CR infarction | 5 † | 1 |
| Other infarction | 3 | 0 |

*: Mean ± standard deviation (year).
†: $p = 0.0024$ (Fisher's exact test).
ICA: internal carotid artery, MCA: middle cerebral artery, TIA: transient ischemic attack, MR: magnetic resonance, CR: corona radiata.

**Table 2** Quantitative measurements of CBF and CVR in the cerebellum

| | affected side | normal side |
|---|---|---|
| CBF* | | |
| without CCD | 41.2 ± 5.6 | 41.7 ± 4.9 |
| with CCD | 36.4 ± 8.6† | 44.0 ± 8.1 |
| CVR | | |
| without CCD | 0.39 ± 0.15 | 0.49 ± 0.3 |
| with CCD | 0.55 ± 0.2 | 0.60 ± 0.3 |

*: Mean ± standard deviation (ml/100 g/min).
†: $p < 0.05$ between affected side and normal side.
CCD: crossed cerebellar diaschisis, CVR: cerebrovascular reserve capacity, CBF: cerebral blood flow.

sides were compared in the patients with CCD and without CCD. Statistical analysis used Student's t-test and Fisher's exact test. A p value of less than 0.05 was considered as statistically significant.

## RESULTS

*Characteristics of patients*

Eighteen of the 30 patients (60%) had CCD (Table 1). Patients with and without CCD showed no significant differences in age, sex or the site of occlusion. Thirteen patients in the CCD group (72%) had minor stroke or transient ischemic attacks, whereas only 5 patients (42%) without CCD had these symptoms. This difference was not statistically significant (Fisher's exact test; $p = 0.1362$). MR imaging evidence of cerebral infarction was detected in 15 patients (83%) with CCD, but in only 3 patients (25%) without CCD. This difference was statistically significant (Fisher's exact test; $p = 0.0024$).

**Table 3** CBF and CVR values in the cerebrum in regions of interest of patients with and without CCD

| | affected side | normal side |
|---|---|---|
| CBF* | | |
| With CCD | | |
| Hemisphere | 28.7 ± 4.3 | 29.1 ± 5.4 |
| Frontal | 40.6 ± 8.4 | 40.9 ± 7.6 |
| Parietal | 34.7 ± 5.9 | 35.5 ± 5.7 |
| Temporo-parietal | 33.7 ± 7.3 | 36.9 ± 6.4 |
| Without CCD | | |
| Hemisphere | 27.5 ± 7.4† | 31.4 ± 6.8 |
| Frontal | 39.1 ± 12.3† | 44.6 ± 9.8 |
| Parietal | 33.8 ± 10.6† | 42.1 ± 11.7 |
| Temporo-parietal | 35.8 ± 11.3† | 40.8 ± 10.4 |
| CVR | | |
| With CCD | | |
| Hemisphere | 0.53 ± 0.22 | 0.55 ± 0.2 |
| Frontal | 0.43 ± 0.28 | 0.48 ± 0.34 |
| Parietal | 0.50 ± 0.18 | 0.47 ± 0.16 |
| Temporo-parietal | 0.44 ± 0.25 | 0.49 ± 0.26 |
| Without CCD | | |
| Hemisphere | 0.42 ± 0.18‡,§ | 0.56 ± 0.38 |
| Frontal | 0.33 ± 0.15‡,§ | 0.47 ± 0.26 |
| Parietal | 0.41 ± 0.11‡,§ | 0.48 ± 0.12 |
| Temporo-parietal | 0.35 ± 0.16‡,§ | 0.50 ± 0.14 |

*: Mean ± standard deviation (ml/100 g/min).
†: $p < 0.05$, ‡: $p < 0.01$ between affected and normal.
§: $p < 0.01$ between with and without CCD.
CBF: cerebral blood flow, CVR: cerebrovascular reserve capacity, CCD: crossed cerebellar diaschisis.

*Quantitative analysis of rCBF*

Cerebellar CBF in patients with CCD was reduced by about 17% on the affected side compared to the normal side, but the CVR showed no significant difference between the affected and normal sides (Table 2). Patients with CCD had no significant difference in site of occlusion or presence of infarction between the affected and normal sides.

The quantitative CCD was significantly correlated with the degree of CVR in all supratentorial regions (temporo-parietal: $r = 0.889$, $p < 0.0001$; hemisphere: $r = 0.868$, $p < 0.0001$; parietal: $r = 0.841$, $p < 0.0001$; frontal: $r = 0.710$, $p = 0.002$).

Measurement of rCBF values in the ROIs in the supratentorial regions showed no significant difference between the affected and normal sides in any ROI in the patients with CCD, but a significant difference in all ROIs in the patients without CCD. There was no significant difference between the patients with and without CCD in

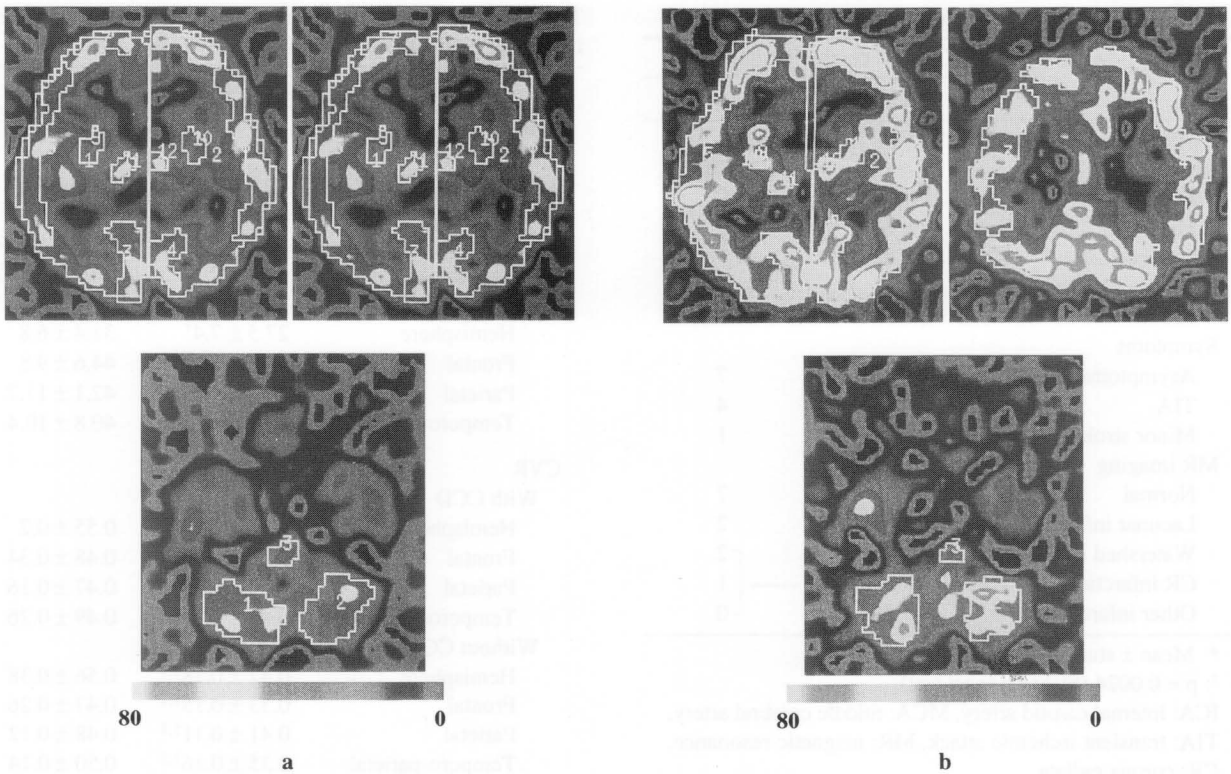


**Fig. 2** A 28-year-old patient with right ICA occlusion. a: The SPECT images at rest show hypoperfusion in the right temporo-parietal lobe and parietal lobe (34.1 ml/100 g/min, 36.2 ml/100 g/min) with crossed cerebellar diaschisis (right 45.7 ml/100 g/min, left 38.3 ml/100 g/min). b: The SPECT images during Diamox loading show a good response in each ROI and no asymmetry in the cerebellum (temporo-parietal lobe: 67.9 ml/100 g/min, parietal lobe: 71.3 ml/100 g/min, right cerebellum: 60.2 ml/100 g/min, left cerebellum: 68.1 ml/100 g/min).

the CBF values in the affected side (Table 3).

Measurement of the CVR in the ROIs in the supratentorial regions revealed no significant difference between the affected and normal sides in any ROI in the patients with CCD, but a significant difference in the patients without CCD. There was a significant difference between the patients with and without CCD (Table 3) in the CVR on the affected side in the ROIs of the cerebral hemispheres, frontal lobe, parietal lobe, and temporo-parietal lobe.

Representative cases are shown in Figures 2 and 3.

## DISCUSSION

The present study showed that the occurrence of CCD was closely related to MR imaging evidence of infarction. The quantitative CCD was 17%, and the CVR was preserved in the cerebellum of patients with CCD. The quantitative CCD was significantly correlated with the degree of CVR in all ROIs. There were significant differences in CBF and CVR between the affected and normal sides in all ROIs of the cerebrum in patients without CCD.

Semiquantitative analysis with $^{123}$I-*N,N,N'*-trimethyl-*N'*-(2-hydroxy-3-methyl-5-iodobenzyl)-1,3-propanediamine 2-HCl SPECT disclosed that CCD was present in 50% of patients with complete stroke and in 24% of patients with reversible ischemic attack.[11] CCD was significantly correlated with the clinical severity and extension of the supratentorial lesion, but CCD was also present in 3 of 16 patients with normal neurological examination and CT findings. Consequently, a 'functional' hemispheric disturbance may be sufficient to produce a remote effect in the contralateral cerebellar hemisphere.[11] Xenon-133 ($^{133}$Xe) inhalation and dynamic SPECT in 11 patients with stroke and large, unilateral cerebral hemispheric infarcts without evidence of cerebellar infarction revealed that cerebellar vasoreactivity is intact in stroke patients with CCD.[27] PET study of carbon dioxide responsiveness to cerebellar blood flow in a patient with major cerebral artery occlusion revealed that the percentage change in cerebellar blood flow per millimeter of mercury $PaCO_2$ change was uniform across the sides affected and unaffected by CCD.[28]

A multiple trial concluded that the rCBF measured by the I-123 IMP SPECT method is significantly correlated with the rCBF obtained by [$^{15}$O]$H_2O$ PET, $^{133}$Xe-SPECT, and I-123 IMP microspheres in a variety of clinical settings.[21] Examination of normal rCBF values and the

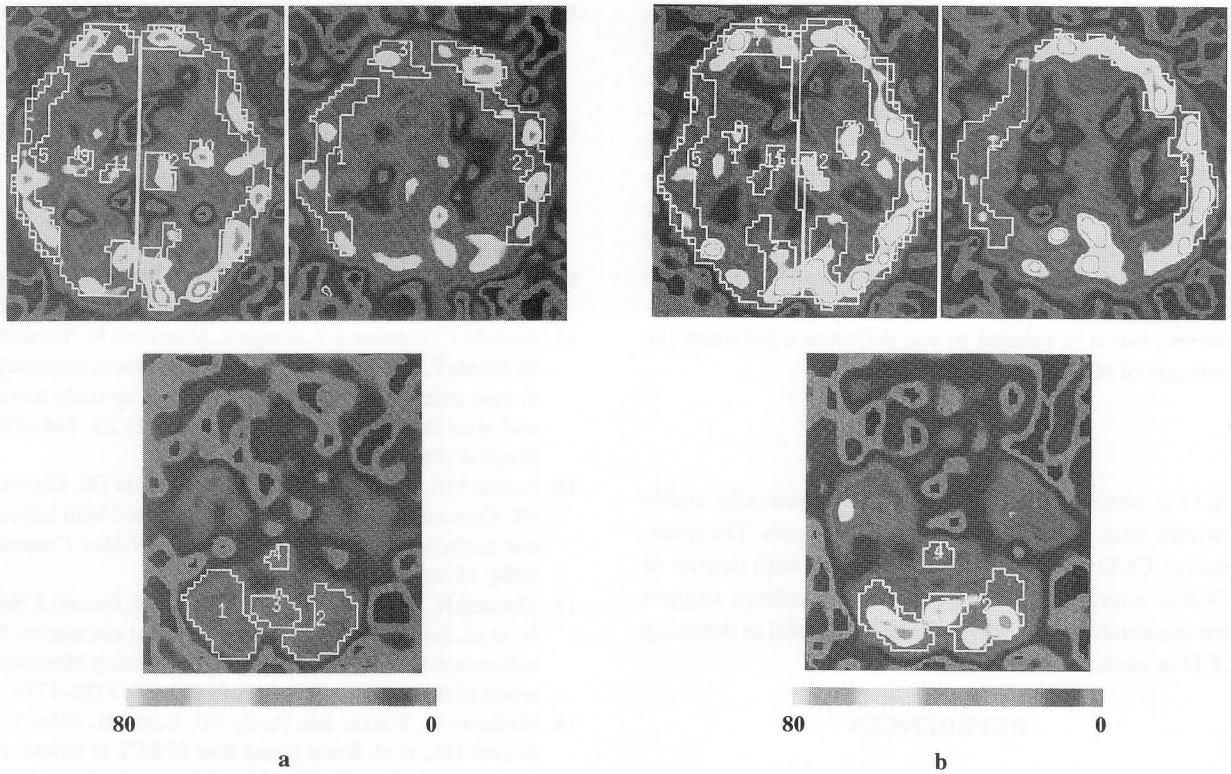


**Fig. 3** A 65-year-old patient with right MCA occlusion. a: The SPECT images at rest showing hypoperfusion in the right hemisphere, temporo-parietal lobe and parietal lobe (35.7 ml/100 g/min, 37.3 ml/100 g/min, 39.3 ml/100 g/min) without crossed cerebellar diaschisis (right 49.3 ml/100 g/min, left 50.9 ml/100 g/min). b: The SPECT images during Diamox loading show poor response in the right hemisphere, temporo-parietal lobe and parietal lobe (40.5 ml/100 g/min, 38.1 ml/100 g/min, 32.3 ml/100 g/min), but good response in the cerebellum (right 79.7 ml/100 g/min, left 80.1 ml/100 g/min).

reproducibility and sensitivity to hypoperfusion in stroke patients also found that the mean rCBF value in the cerebral cortex was 33.0 ± 5.1 ml/100 g/min.[22] The whole-brain CBF values showed high reproducibility, with high correlations between values obtained at the first and second studies, indicating that the I-123 IMP method is reproducible, sensitive to hypoperfusion, and allows quantitative evaluation of rCBF in routine clinical practice.[22]

Previous quantitative analysis of CCD in patients with major cerebral artery occlusion has used PET and SPECT.[12,13] PET measurement of rCBF, oxygen metabolism, oxygen extraction fraction (OEF), and cerebral blood volume (CBV) in the cerebral and cerebellar cortices of 15 patients with unilateral major cerebral artery occlusive disorders showed that 9 patients had crossed cerebellar hypoperfusion (CCH) and 6 had no such hypoperfusion. Evaluation of absolute hemispheric values showed that patients without CCH had decreased CBF, increased OEF, and decreased CBF/CBV in the affected cerebral cortex compared to patients with CCH. A SPECT study with technetium-99m-hexamethylpropyleneamine oxime ($^{99m}$Tc-HMPAO) in 14 patients with major cerebral occlusive or stenotic disorders found only 6 patients with CCD and no difference in the CBF ratio (MCA territory only) between patients with and without CCD. The CBF ratio after acetazolamide stress was significantly higher in the patients with CCD than those without CCD and the difference in the CBF ratio before and after acetazolamide stress correlated significantly with the degree of CCD ($r = -0.794$, $p < 0.01$).[13] Our results agree with the PET study, and although OEF is not equivalent to CVR, a close relationship was recognized between these two findings.

The present study showed that the normal rCBF and CVR values in the cerebral cortex were 35.6 ± 3.2 ml/100 g/min and 0.51 ± 0.09, and detected a reduction in the CBF and CVR in every ROI on the affected side in patients without CCD. The previous two studies examined only one ROI, such as the hemisphere or MCA territory.[12,13] The SPECT study with $^{99m}$Tc-HMPAO agrees with our CVR findings, but no CBF reduction was observed. This difference depends on the severity of hemodynamic compromise in the patients examined and the differences in the radiopharmaceuticals. The PET study[12] also disclosed that the quantitative CCD was significantly correlated with the difference in oxygen metabolism on the affected and normal sides. Although SPECT cannot measure the

oxygen metabolism directly, detecting CCD may help in detecting functional abnormality in the supratentorial region. The causative factor of CCD remains controversial. Damage to corticopontocerebellar, cerebelloruburothalamic, and thalamocortical tracts may result in CCD.[1,2] Our finding CCD even on the affected side with a reduction in rCBF was controversial, but this may be due to the damage to several tracts, and another controversial point was that rCBF on the affected side in those without CCD was significantly decreased, and the reason is unknown, but it is related to the difference between the methods of rCBF measurement.

## CONCLUSION

CCD is observed frequently even in clinically stable patients with major cerebral artery occlusion. The quantitative CCD is significantly correlated with degree of CVR in supratentorial regions. These findings suggest that quantitative I-123 IMP SPECT is helpful in detecting CCD in such patients.

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