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Crossed Cerebellar and Cerebral Cortical Diaschisis in Basal Ganglia Hemorrhage

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Abstract

Purpose: The purpose of this study was to evaluate the phenomenon of diaschisis in the cerebellum and cerebral cortex in patients with pure basal ganglia hemorrhage using cerebral blood flow SPECT. **Materials and Methods:** Twelve patients with pure basal ganglia hemorrhage were studied with Tc-99m ECD brain SPECT. Asymmetric index (AI) was calculated in the cerebellum and cerebral cortical regions as $|C_R-C_L|$ $/(C_R-C_L)\times 200$, where C_R and C_L are the mean reconstructed counts for the right and left ROIs, respectively. Hypoperfusion was considered to be present when AI was greater than mean+2 SD of 20 control subjects. **Results:** Mean AI of the cerebellum and cerebral cortical regions in patients with pure basal ganglia hemorrhage was significantly higher than normal controls (p<0.05): Cerebellum (18.68±8.94 vs 4.35±0.94, mean±SD), thalamus (31.91±10.61 vs 2.57±1.45), basal ganglia (35.94±16.15 vs 4.34±2.08), parietal (18.94±10.69 vs 3.24±0.87), frontal (13.60±10.8 vs 4.02±2.04) and temporal cortex (18.92±11.95 vs 5.13±1.69). Ten of the 12 patients had significant hypoperfusion in the contralateral cerebellum. Hypoperfusion was also shown in the ipsilateral thalamus (n=12), ipsilateral parietal (n=12), frontal (n=6) and temporal cortex (n=10). **Conclusion:** Crossed cerebellar diaschisis (CCD) and cortical diaschisis may frequently occur in patients with pure basal ganglia hemorrhage, suggesting that CCD can develop without the interruption of corticopontocerebellar pathway. **(Korean J Nucl Med 1998;32:397-402)**

Key Words: Crossed cerebellar diaschisis, Cortical diaschisis, Basal ganglia hemorrhage, Brain SPECT

Introduction

Diaschisis refers to functional deactivation oc-

curring remotely from the responsible structural lesion. It has been suggested that this phenomenon results from an interruption of afferent or efferent fiber pathways.[1-4] Crossed cerebellar diaschisis (CCD), a matched depression of blood flow and metabolism in the cerebellar hemisphere contralateral to a focal supratentorial lesion, is a well recognized phenomenon following cerebral infarction.[1,2,5-8]

It has been described that interruption of the corticopontocerebellar tract is the most likely mechanism of CCD. However, a few reports have described CCD after hemorrhage in subcortical structures such as basal ganglia and thalamus, which may not be directly connected to the corticopontocerebellar tract.[9,10] In addition, the remote effect has been observed not only in the contralateral cerebellum, but also in the cerebral cortex in patients with recent ischemic or hemorrhagic unilateral capsulothalamolenticular lesions sparing the cortex.[11]

The purpose of this study was to evaluate the phenomenon of diaschisis in the cerebellum and cerebral cortex in patients with pure basal ganglia hemorrhage using cerebral blood flow SPECT.

Materials and Methods

1. Patients

This study included 12 patients with hypertensive intracerebral hemorrhage strictly confined to basal ganglia on initial and follow-up CT/MRI. There were 8 men and 4 women ranging in age from 30 to 67 year with a mean age of 50.3 ± 10.2 year. None of the patients had structural abnormalities in the cerebellum, cerebral cortex and internal capsule on CT/MRI. Patients who had clinical symptoms of ischemic episode before hemorrhagic attack and/or MR findings suggesting previous ischemic episode were excluded. None of the patients had a second symptomatic neurologic event since the hemorrhagic attack. The mean interval from onset of symptoms to the SPECT examination was 58.17 ± 30.73 days. The patients' clinical characteristics are summarized in Table 1.

SPECT studies were also performed in 20 patients with psychiatric problems who had no prior history of neurological deficits or vascular risk factor (12 men and 8 women, age 43.5 ± 11.4 year) as control group. All of the subjects had normal SPECT and MRI scans.

2. Imaging procedures

SPECT was performed after an intravenous injection of 740 MBq of Tc-99m ethyl cysteinate dimer (ECD) using a brain-dedicated annular crystal gamma camera (Digital Scintigraphic Inc,

Table 1. Patient Profiles and Calculated Asymmetric Index in Each Region

Sex/Age	Interval*	Calculated asymmetric index in each region					
		Cerebellum	Basal ganglia	Thalamus	Parietal	Frontal	Temporal
M/30	42	21.2	34.4	49.6	37.1	26.9	21.7
F/54	45	15.4	16.1	30.5	30.7	2.2	27.3
M/44	34	17.7	35.5	18.3	9.2	3.7	19.2
F/59	87	17.1	70.4	39.9	8	7.3	5.8
M/67	99	19.9	39.1	22.8	23.4	16	19.4
M/53	21	23.3	51.7	45.2	25	5.4	14.4
M/59	108	14.2	23.2	21.9	7.12	10.6	1.2
F/46	44	23.1	23.9	41.5	26.7	33.4	47
M/37	95	30.5	55.6	37.8	28.9	28.8	27.9
M/56	22	33.8	19	30.7	10.4	5.4	20.2
M/47	47	4.6	32.8	20.5	11.4	17.3	10.5

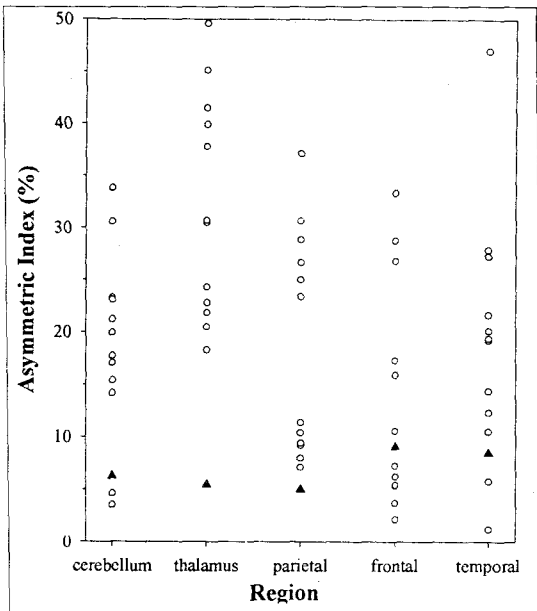*Interval: duration (day) between the onset of hemorrhagic attack and SPECT study.

Fig. 1. Scatter plot of AI values of patients in each region. AI values for patients are shown as open circles. Closed triangles represent control mean AI+2 SD in each region. Open circles above the closed triangle indicate significant hypoperfusion.

Waltham, USA) with low-energy high resolution parallel hole collimators. One hundred twenty projections were acquired with 3-degree angular increments. The matrix size was 128×128. Transaxial images were reconstructed by filtered backprojection using a Butterworth filter (Nyquist frequency 1.1 cycle/cm at an order No. 10). Attenuation correction was performed by Chang's method.

3. Data analysis

Three or four transaxial slices parallel to orbito-meatal line were selected for typical brain level according to an human brain atlas, delineating ana-tomic structures at the different slice levels. On these transaxial SPECT images, regions of interest (ROIs) were drawn over the thalamus, basal gan-glia, cerebellum, and frontal, parietal and temporal cortices. The size of each ROI could be manually

adjusted, so that its contour fit the each structue well. Asymmetric index (AI) was calculated in each region as $|C_R - C_L| / (C_R + C_L) \times 200$, where C_R and C_L are the mean reconstructed counts for the right and left ROIs, respectively.[7] We defined that hypoperfusion was evident when AI was greater than control mean+2 SD.

Results

In the control group, mean AI was 4.35 ± 0.94 (mean$\pm$SD) in cerebellum, 2.57 ± 1.45 in thala-mus, 4.34 ± 2.08 in basal ganglia, 3.24 ± 0.87 in parietal, 4.02 ± 2.04 in frontal, and 5.13 ± 1.69 in temporal cortcies. Mean AI of patients with pure basal ganglia hemorrage was 18.68 ± 8.94 in cere-bellum, 31.91 ± 10.61 in thalamus, 35.94 ± 16.15 in basal ganglia, 18.94 ± 10.69 in parietal, 13.60 ± 10.8 in frontal, and 18.92 ± 11.95 in temporal cor-tex, all of which were significantly higher than that of the control group (p<0.05)(Table 1).

Figure 1 shows the AI values of patients in each region. Ten of the 12 patients had significant hypo-perfusion in the contralateral cerebellum (i.e., AI>control mean+2 SD). Hypoperfusion was also shown in the ipsilateral thalamus (n=12), ipsilateral parietal (n=12), frontal (n=6) and temporal (n=10) cortices. Representative SPECT images are shown in Figure 2.

Discussion

The mechanism underlying CCD has been ascri-bed to the disruption of the corticopontocerebellar pathway, which is cerebellar afferents from the pons to cerebellum via the middle cerebellar pe-duncle. While there has been many reports on CCD after cerebral cortical infarction,[1,2,5-8] only a few studies reported CCD in patients with the deep seated infarction such as in thalamus.[4,10] Pappata,
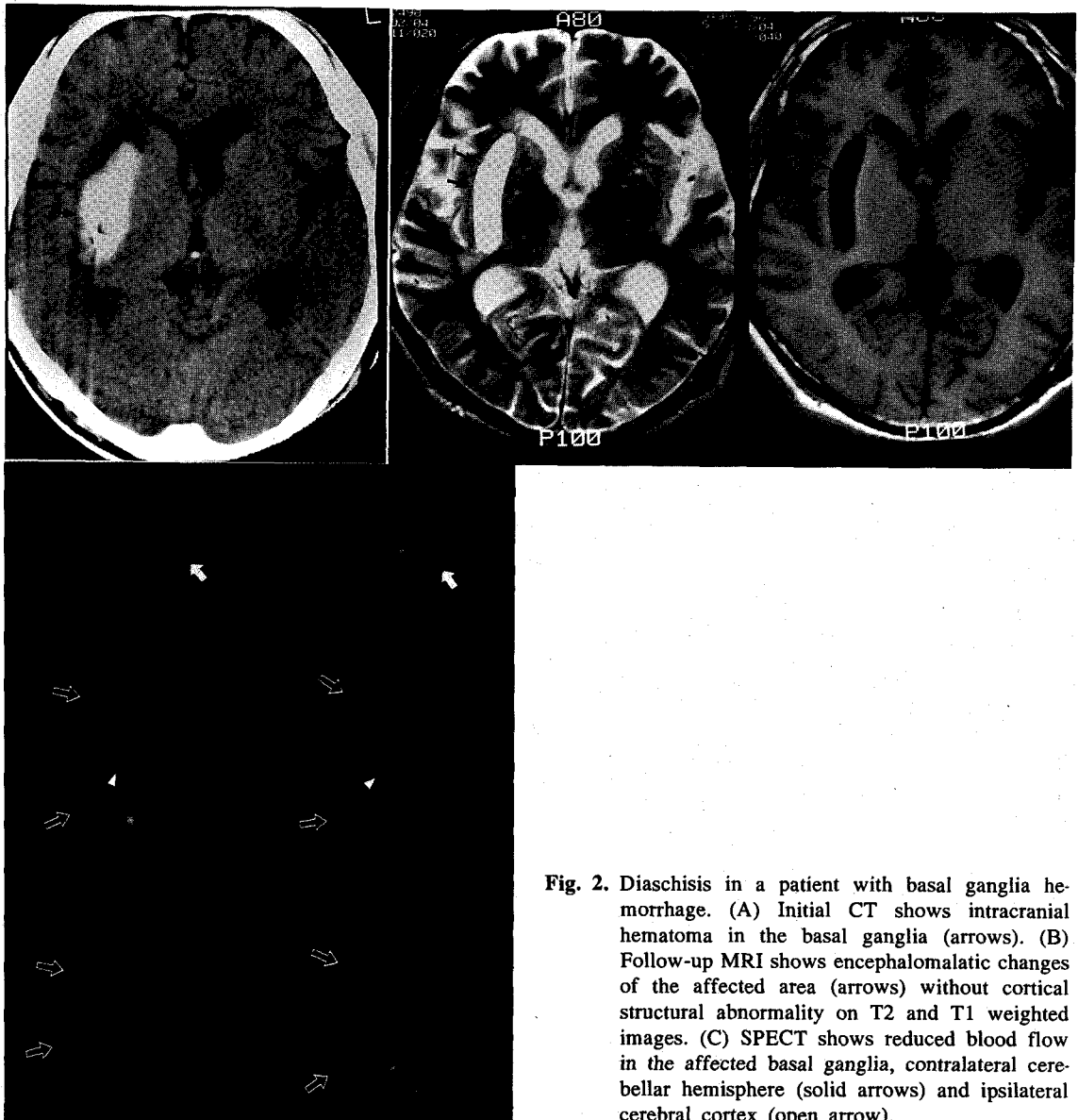
Fig. 2. Diaschisis in a patient with basal ganglia hemorrhage. (A) Initial CT shows intracranial hematoma in the basal ganglia (arrows). (B) Follow-up MRI shows encephalomalatic changes of the affected area (arrows) without cortical structural abnormality on T2 and T1 weighted images. (C) SPECT shows reduced blood flow in the affected basal ganglia, contralateral cerebellar hemisphere (solid arrows) and ipsilateral cerebral cortex (open arrow).

et al. reported that CCD was found in two of six patients with thalamic infarction.[10] They explained that this phenomenon may have resulted either from the damage to the cerebellar efferent pathway, (i.e. ascending cerebellothalamocortical system) or indirectly from the hypofunction of the cerebral cortex. Postmortem studies, which have shown that thalamic lesions may result in retro-

grade contralateral dentate nucleus atrophy, may support this hypothesis.[12]

In our study, brain SPECT of patients with pure basal ganglia hemorrhage frequently showed decreased blood flow in remote areas such as the ipsilateral cerebral cortex and the contralateral cerebellum. Also, Haruyuki et al. have described that putaminal hemorrhage may result in CCD.[9]

On the basis of anatomical connections between basal ganglia and cerebellum, there are at least three putative pathways which could be involved in this phenomenon. First, basal ganglia have many neuronal connections with thalamus. The striatum (caudate and putamen) receives inputs from the intralaminar thalamic nuclei and gives inhibitory axons (GABAnergic) to the globus pallidus, which is the major outflow nucleus of the corpus striatum. The globus pallidus, in turn, gives inhibitory axons to the ventral nuclei (ventral anterior and ventral lateral) of the thalamus which also receives input from the cerebellum (cerebellar efferent pathway).[13] And then, the interruption of this circuit in the region of basal ganglia is assumed to be responsible for the reduction of blood flow in the contralateral cerebellar hemisphere via cerebellar efferent pathway. Second, basal ganglia also have many neuronal connections with cerebral cortex.[14] Our study showed a significant hypoperfusion in ipsilateral cortex in all patients. Hence, CCD in patients with basal ganglia hemorrhage may be resulted indirectly from the hypoperfusion of cerebral cortex. Finally, an anatomical neurochemical pathway (dopaminergic pathway) arises from dentate nucleus of cerebellum. It crosses the midline at the level of the brachium conjunctivum and send terminals to substantia nigra. The projections from the substantia nigra enter the neostriatum.[15,16] Therefore, interruption of this neuronal circuit may be another possible explanation of CCD in patients with basal ganglia hemorrhage.

In conclusion, crossed cerebellar diaschisis and cortical diaschisis may frequently occur in patients with pure basal ganglia hemorrhage. These data suggest that CCD can be developed without the interruption of corticopontocerebellar pathway.

요   약

목적: 대뇌피질의 구조적 병변이 없는 기저핵 혈종 환자에 있어서, 대뇌와 소뇌의 해리 현상에 관해 알아보고자 하였다. 대상 및 방법: CT와 MRI상 혈종이 기저핵에 국한되고, 대뇌 피질의 구조적 병변이 없는 12명의 환자를 대상으로 하였다. Tc-99m ECD SPECT검사가 전 예에서 시행되었고, 대조군으로 MRI상 구조적 병변이 없고 SPECT검사상 육안적으로 정상 소견을 보인 20명의 정신과 환자가 선택되었다. 해리 현상에 의한 rCBF의 변화를 육안적으로 확인하였고, 반정량적 분석을 위해 asymmetric index (AI)를 이용하였다. AI가 기저핵, 시상, 소뇌와 대뇌 피질 (전두엽, 측두엽, 두정엽)에서 측정되었고, 대조군 AI의 (평균+2×표준편차)보다 큰 AI를 가지는 경우를 저혈류로 정의하였다. 결과: 환자군에서 측정된 소뇌와 대뇌 피질의 평균 AI는 대조군과 비교하여 통계학적으로 의미 있게 큰 수치를 보였다 (p<0.05): 소뇌 (18.688.94 vs 4.35 0.94, 평균 표준표차), 시상 (31.9110.61 vs 2.57 1.45), 기저핵 (35.9416.15 vs 4.34 2.08), 두정엽 (18.9410.69 vs 3.24 0.87), 전두엽 (13.6010.8 vs 4.02 2.04), 측두엽 (18.9211.95 vs 5.13 1.69). 12명의 환자 중 혈종의 반대측 소뇌(10명), 동측 시상 (12명) 동측 두정엽 (12명), 전두엽 (6명), 측두엽 (10명)에서 육안적으로 또한 AI를 이용한 반정량적 분석상 의미 있는 저혈류를 보였다. 결론: 교차소뇌해리 현상과 피질 해리 현상은 기저핵 혈종 환자에서 빈번하게 관찰되었다. 이는 교차소뇌해리 현상이 기존에 알려진 피질뇌교소뇌로 (corticopontocerebellar tract)의 장애가 없이도 발생할 수 있다는 것을 시사한다.

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