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 \(|C_R-C_L|/(C_R+C_L)\times200\), 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 occurring 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.\(^{1,2,5,4}\)
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. 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.  

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* | 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.
adjusted, so that its contour fit the each structure well. Asymmetric index (AI) was calculated in each region as \( \frac{|C_R - C_L|}{(C_R + C_L)} \times 200 \), where \( C_R \) and \( C_L \) are the mean reconstructed counts for the right and left ROIs, respectively. 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 ± SD) in cerebellum, 2.57 ± 1.45 in thalamus, 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 cortices. Mean AI of patients with pure basal ganglia hemorrhage was 18.68 ± 8.94 in cerebellum, 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 cortex, 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 hypoperfusion 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 ascribed to the disruption of the corticopontocerebellar pathway, which is cerebellar afferents from the pons to cerebellum via the middle cerebellar peduncle. While there has been many reports on CCD after cerebral cortical infarction, only a few studies reported CCD in patients with the deep seated infarction such as in thalamus.
et al. reported that CCD was found in two of six patients with thalamic infarction.\textsuperscript{10} They explained that this phenomenon may have resulted either from the damage to the cerebellar efferent pathway, (i.e. ascending cerebellothalamiccortical system) or indirectly from the hypofunction of the cerebral cortex. Postmortem studies, which have shown that thalamic lesions may result in retrograde contralateral dentate nucleus atrophy, may support this hypothesis.\textsuperscript{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.\textsuperscript{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 (GABAergic) 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). 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. 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. 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.

References


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