Crossed Cerebellar Diaschisis Detected by Arterial Spin-Labeled Perfusion Magnetic Resonance Imaging in Subacute Ischemic Stroke

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Background: Crossed cerebellar diaschisis (CCD) was a common radiological phenomenon manifested as reduced blood flow and metabolism in the cerebellar hemisphere contralateral to a supratentorial cerebral lesion. The hypoperfusion and hypometabolism in the contralateral cerebellum in CCD was traditionally detected by positron emission tomography (PET) and single-photon emission computed tomography (SPECT). The present prospective study aimed to assess the detection of CCD in subacute stage ischemic stroke by arterial spin-labeling (ASL) perfusion technique with a 3.0-T magnetic resonance imaging (MRI) scanner. *Methods:* ASL images were obtained from 46 patients with supratentorial ischemic stroke at subacute stage. Regional cerebral blood flow values in the cerebellar hemispheres were measured on a region of interest basis. *Results:* Twenty-four of 46 (52%) patients showed CCD phenomenon by ASL-MRI method, which was in line with the PET/SPECT series. Infarctions in basal ganglia areas are prone to cause CCD. *Conclusions:* With advantages in easy acquisition and no radiation, ASL-MRI seems to be an ideal tool for the detection and follow-up of CCD. **Key Words:** Crossed cerebellar diaschisis—arterial spin-labeling—magnetic resonance imaging—stroke.

Introduction

Crossed cerebellar diaschisis (CCD) is a common radiological phenomenon manifested as reduced blood flow and glucose metabolism in the cerebellar hemisphere contralateral to a supratentorial cerebral lesion.[1,2] Although mostly seen in cerebral stroke, CCD has been reported in other clinical conditions such as status epilepticus, glioma, encephalitis, and cerebral ischemia–reperfusion process.[3-6] CCD can be long lasting as seen in many ischemic stroke cases or transient and reversible as seen in status epilepticus. It is considered as a continuum between potentially reversible hypoperfusion status and irreversible degeneration depending on the reversal of abnormal supratentorial cerebral lesion. The interruption of the cerebropontine-cerebellar pathway is thought to be the underlying cause of CCD.

Originally considered as merely a neuroradiological phenomenon, recent findings suggested CCD as an important prognostic indicator of stroke recovery and treatment response.[7-9] There is also increasing evidence

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for the cerebellar involvement in cognitive function notably in executive control, verbal memory, and language.[10] A hypothesis that disrupted cerebellar function by CCD after stroke may result in vascular cognitive impairment in the long run has been conceived.[11] Thus, it is necessary to find an easy, noninvasive, and widely available imaging method for the detection and intensive study of CCD.

The hypoperfusion and hypometabolism in the contralateral cerebellum in CCD was traditionally detected by positron emission tomography (PET) or single-photon emission computed tomography (SPECT). A study using perfusion-weighted magnetic resonance imaging (PWI) found PWI could detect CCD but with a reduced sensitivity compared with the previous findings by PET/SPECT.[12] Another similar study found 1.5 T PWI-MRI was not suited to detect CCD after stroke because of poor accuracy in comparison with PET.[13]

Arterial spin labeling (ASL) is a novel noninvasive MRI-based technique for quantitative cerebral blood flow (CBF) measurement. Briefly, ASL-MRI takes advantage of magnetically labeled blood water as an endogenous tracer for quantification of brain perfusion. Unlike PET and SPECT, ASL-MRI was more available and less expensive without exposure to ionizing radiation. These make ASL perfusion technique being rigorously explored in many different disease conditions and has gained success in stroke and neurodegenerative conditions.[14]

Considering the previously mentioned evidence, the present study aimed to assess the detection of CCD in subacute stage ischemic stroke by ASL-MRI.

Materials and Methods

Subjects

Forty-six patients (34 male and 12 female) with their first-ever acute ischemic stroke were enrolled by the Department of Neurology in People's Hospital of Zhengzhou University. On admission, the diagnosis of acute hemispheric ischemic stroke was made according to the symptoms and signs and MRI findings (series: T1WI, T2WI, diffusion weighted imaging [DWI], fluid attenuated inversion recovery [FLAIR], magnetic resonance angiography [MRA]). Patients who met the following criteria received the follow-up MRI examinations for the detection of CCD (series: T1WI, DWI, ASL): (1) a unilateral supratentorial DWI lesion; (2) no supratentorial chronic stage stroke, no infarct in the brain stem or cerebellum, no cerebral tumor, cerebral trauma, or other concomitant critical illness present on DWI or T2WI scans; (3) MRA showing no significant stenosis of vertebral basilar artery and the major branches; and (4) the patients were likely to undergo the follow-up MRI examinations during the subacute stage (1-3 weeks after stroke onset).

The mean age of the patients was 56.89 ± 10.42 years. The time that the patient was last known to be healthy was recorded as the time of stroke onset. The mean time from the onset of stroke to ASL perfusion examination was 10.87 ± 4.28 days. The institutional ethics committees approved this study, and informed consents for the participation in the study were obtained from each patient or the patient's relative.

Image Acquisition and Processing

Magnetic resonance acquisition was performed on a 3.0-T magnetic resonance scanner (GE Discovery 750, Milwaukee, WI) using a standard 8-channel quadrature transmit–receive head coil. Sequences obtained were conventional T2WI, DWI, MRA, and ASL. DWI was performed with a b-value of 1000 seconds/mm^2, repetition time (TR) = 6000 ms, field of view = 24 cm, matrix = 128 × 128, slice thickness = 5 mm, skip = 1.5 mm, slices = 20, number of excitations (NEX) = 2, gradients in 3 tetrahedrally encoded directions to create isotropic DWI, and apparent diffusion coefficient maps. Pseudocontinuous ASL perfusion images were collected using 3D fast spin echo acquisition with background suppression, with postlabeling delay of 1525 ms. TR = 4601 ms, TE = 10.5 ms, field of view = 24 cm, matrix = 128 × 128, NEX = 3, slice thickness = 4 mm, and slices = 38. CBF maps were calculated from the perfusion-weighted images using a 2-compartment model with a finite labeling duration, as described previously.[15,16]

To assess the presence of CCD, circular regions of interest, measuring 25 mm in diameter, were placed in the cerebellar hemispheres ipsilateral (I) and contralateral (C) to the hemispheric stroke. The degree of CCD was measured on one axial cut representing the highest cerebellar asymmetry. From the CBF value, we quantified the degree of hypoperfusion by calculating asymmetry indices for the cerebellar hemisphere according to the following formula: (CBFipsilateral − [CBFcontralateral/CBFipsilateral] × 100%). CCD was considered present when asymmetry indice was less than .1. To investigate relationships between the location of supratentorial infarcts and CCD, the ischemic lesions on DWI were classified by 8 main anatomic regions: frontal lobe, parietal lobe, temporal lobe, occipital lobe, basal ganglia, corona radiata, thalamus and limbic system, or insular lobe.

Statistical Analysis

Statistical analysis was performed with SPSS 18.0 (IBM Inc, Armonk, NY). Demographic data from the CCD positive [CCD(+)] and CCD negative [CCD(−)] patients were examined by the independent samples t test and chi-square test. Association of CCD and infarction locations in different supratentorial brain regions was determined by the chi-square or Fisher exact test if it is more appropriate. A 2-tailed P value less than .05 was considered significant.