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Magnetic resonance perfusion imaging evaluation in perfusion abnormalities of the cerebellum after supratentorial unilateral hyperacute cerebral infarction★

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Abstract

Magnetic resonance imaging (MRI) data of 10 patients with hyperacute cerebral infarction (≤ 6 hours) were retrospectively analyzed. Six patients exhibited perfusion defects on negative enhancement integral maps, four patients exhibited perfusion differences in pseudo-color on mean time to enhance maps, and three patients exhibited perfusion differences in pseudo-color on time to minimum maps. Dynamic susceptibility contrast-enhanced perfusion weighted imaging revealed a significant increase in region negative enhancement integral in the affected hemisphere of patients with cerebral infarction. The results suggest that dynamic susceptibility contrast-enhanced perfusion weighted imaging can clearly detect perfusion abnormalities in the cerebellum after unilateral hyperacute cerebral infarction.

Key Words: magnetic resonance imaging; magnetic resonance-perfusion-weighted imaging; cerebral infarction; cerebral perfusion; functional neurological deficit

Abbreviation: DSC-PWI, dynamic susceptibility contrast-enhanced perfusion weighted imaging; rNEI, region negative enhancement integral; rMTE, region mean time to enhance; rTTM, region time to minimum

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INTRODUCTION

In recent years, with the rapid development of magnetic resonance (MR) software and hardware technology, the diagnostic value of dynamic susceptibility contrast-enhanced perfusion weighted imaging (DSC-PWI) in cases of cerebral infarction is increasingly widely recognized[1-3]. This technique has been used to assess ischemic penumbra[4-6] and to study the reserve capacity of ischemic brain tissue[7]. Stroke can cause functional inhibition of the contralateral cerebellar hemisphere, leading to abnormal hypoperfusion[8], also referred to as crossed cerebellar diaschisis. Numerous studies have reported that crossed cerebellar diaschisis always occurs after supratentorial infarction[8-11]. However, functional inhibition of the contralateral cerebellum is often concealed by symptoms of cerebral infarction. A previous study demonstrated that patients with cerebral infarction exhibited overlap of the damage related network and the rehabilitation-related network[12]. In addition, it was found that motor function recovery could be promoted by hematogenously brain tissue function after cerebral infarction[12]. These findings indicate that diaschisis may play a key role in the recovery of patients with cerebral infarction. The contralateral cerebellar hypoperfusion was considered to indicate a worse clinical outcome after supratentorial infarct[13]. Cerebral hypoperfusion in the contralateral cerebellar hemisphere after subacute stroke has reported by a number of single-photon emission computed tomography (SPECT) and positron-emission tomography (PET) studies[9-10, 14-15]. However, the application of PET/SPECT equipment is limited by inconvenience, the high cost of equipment and scanning, and the involvement of radiation[16-17]. Only a few studies have used PWI to examine cerebral hypoperfusion in the contralateral cerebellar hemisphere after subacute stroke[8, 11], and no studies have been undertaken in the Chinese population. The current study sought to determine the diagnostic value of DSC-PWI scans in the evaluation of cerebral hypoperfusion in the contralateral cerebellar hemisphere, by conducting a retrospective review of patients with hyperacute cerebral infarction within 6 hours of symptom onset, detected by 3.0T DSC-PWI.

RESULTS

Quantitative analysis of participants

A total of 17 patients with hyperacute stroke who presented at the First Affiliated Hospital of Wenzhou Medical College were included, from December 2007 to September 2010. Seven patients were excluded because they did not meet the inclusion criteria; three of these patients exhibited abnormal signals in the cerebellar hemispheres on diffusion-weighted imaging; two of them exhibited multiple stenosis of arteries on magnetic resonance angiography; and two of them exhibited susceptibility artifacts from the skull base. The remaining 10 patients were included in the final analysis.

Clinical imaging characteristics in patients with hyperacute cerebral infarction

All 10 patients with hyperacute cerebral infarction showed an area of high signal intensity in the unilateral cerebrum on DWI. Of these, four exhibited middle cerebral artery infarction in dominant hemisphere, and six exhibited middle cerebral artery infarction in the non-dominant hemisphere (Figure 1). None of the patients exhibited abnormal signals on DWI, or vertebrobasilar disease on MR angiography between the right and left cerebellar hemispheres (Figure 1). Clinical data are shown in Table 1, and in supplementary Figures 1–9 online.

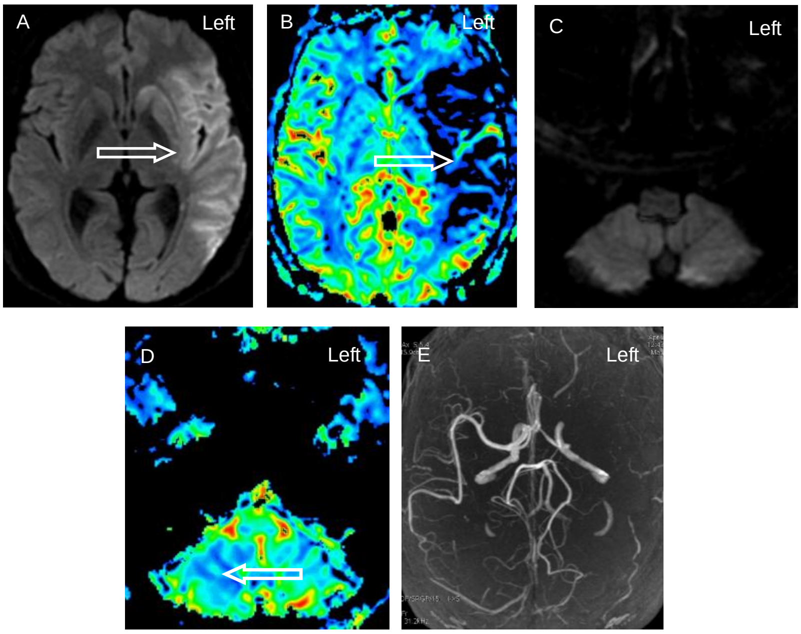

Figure 1 Clinical characteristics of patients with hyperacute cerebral infarction.

(A) Diffusion weighted imaging shows large acute infarct localized in the left frontal lobe top, the basal ganglia (arrow) in 10 cases.

(B) Dynamic susceptibility contrast-enhanced perfusion weighted imaging maps according to region negative enhancement integral exhibiting hypoperfusion (arrow).

(C) Diffusion weighted imaging shows no evidence of infarct in the contralateral cerebellar region.

(D) Dynamic susceptibility contrast-enhanced perfusion weighted imaging maps according to region negative enhancement integral showed contralateral cerebellar hypoperfusion relative to the opposite hemisphere (arrow).

(E) Magnetic resonance angiography maps do not show vertebrobasilar disease.

Table 1 Patients' clinical data

Patient	Gender	Age (year)	Weight (kg)	Height (m)	Onset age (year)	Disease course (hour)	Reasons for unsuitability of CT	Infarction position
1	Male	48	65	1.73	48	2.5	Iodine allergy	Right side
2	Male	43	73	1.69	43	3.0	Family member's unwillingness	Right side
3	Male	73	74	1.75	73	3.2	Family member's unwillingness	Left side
4	Male	71	75	1.76	71	3.0	Family member's unwillingness	Left side
5	Male	64	73	1.72	64	3.1	Family member's unwillingness	Left side
6	Female	54	62	1.63	54	5.2	Family member's unwillingness	Right side
7	Male	49	74	1.71	49	4.7	Family member's unwillingness	Left side
8	Male	71	69	1.78	71	3.5	Family member's unwillingness	Right side
9	Male	54	78	1.73	54	4.5	Iodine allergy	Right side
10	Female	73	72	1.67	73	4.0	Family member's unwillingness	Left side

CT: Computed tomography.

Maps of region negative enhancement integral (rNEI), region mean time to enhance (rMTE) and region time to minimum (rTTM) in patients with hyperacute cerebral infarction

In six patients (60%, 6/10), rNEI maps showed crossed cerebellar hypoperfusion as relatively dark areas in the contralateral cerebellar hemisphere (affected hemisphere) compared with the ipsilateral hemisphere (unaffected hemisphere). When differences in judgment arose between the two examiners, consensus was reached after discussion. Of these patients, four exhibited obvious relative dark areas, and two exhibited mild relative dark areas (Figure 2). In four patients (40%, 4/10), the rMTE maps showed crossed cerebellar perfusion prolonged in the affected hemisphere compared with the unaffected hemisphere. Of these patients, two exhibited obvious relative prolonged perfusion, and two exhibited mild prolonged perfusion. In three patients (30%, 3/10), the rTTM maps showed crossed cerebellar perfusion prolonged in the affected hemisphere compared with the unaffected hemisphere. Of these patients, one exhibited obvious relative prolonged perfusion and three exhibited mild prolonged perfusion (Table 2).

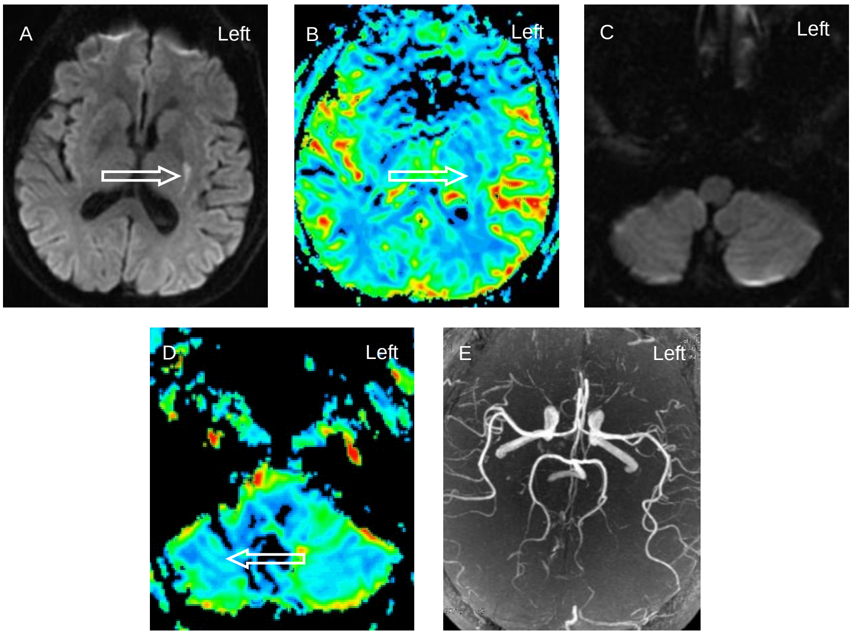

Figure 2 Maps of region negative enhancement integral (rNEI) showed perfusion defects in six patients with hyperacute cerebral infarction.

Diffusion-weighted imaging (DWI) (A) shows small acute infarct localized in the left basal ganglia (arrow); dynamic susceptibility contrast-enhanced perfusion weighted imaging (DSC-PWI) maps according to rNEI show hypoperfusion (B) (arrow).

DWI (C) shows no evidence of infarct in the region of the contralateral cerebellum; DSC-PWI maps according to rNEI show contralateral cerebellar hypoperfusion relative to the opposite hemisphere (D) (arrow). Magnetic resonance angiography (E) maps do not show vertebrobasilar disease.

Table 2 Results of rNEI maps, rMTE maps and rTTM maps in patients with hyperacute cerebral infarction [*n*(%)]

Abnormal signals on the cerebellum	rNEI maps	rMTE maps	rTTM maps
Obvious	4(40)	2(20)	1(10)
Moderate	2(20)	2(20)	2(20)
Mild	4(40)	6(60)	7(70)

Mild: No perfusion defect; moderate: mild perfusion defect; obvious: obvious perfusion defect. rNEI: Region negative enhancement integral; rMTE: region mean time to enhance; rTTM: region time to minimum.

Measurement results of DSC-PWI in patients with hyperacute cerebral infarction

In all 10 patients, the rNEI values of the unaffected cerebellar hemisphere were significantly lower than those of the affected hemisphere ($P < 0.05$). There was no significant difference in the rMTE values and rTTM values between hemispheres ($P > 0.05$; Table 3).

Table 3 Differences in rNEI, rMTE and rTTM of the affected hemisphere relative to the unaffected hemisphere in the cerebellum of patients with hyperacute cerebral infarction

Position	rNEI values	rMTE values	rTTM values
Affected hemisphere	234.6±19.6	139.7±18.0	25.6±9.9
Unaffected hemisphere	199.4±21.0	137.2±9.0	25.3±8.7
t	2.571	1.203	0.602
P	0.037	0.268	0.564

Data were presented as mean ± SD and compared with a two-tailed *t* test assuming unequal variances. rNEI: Region negative enhancement integral; rMTE: region mean time to enhance; rTTM: region time to minimum.

DISCUSSION

The mechanism of cerebral hypoperfusion in the contralateral cerebellar hemisphere after stroke is currently unclear, but may be linked to crossed cerebellar diaschisis[8]. Crossed cerebellar diaschisis results from an interruption of fibers in the cerebellar hemisphere contralateral to a supratentorial stroke, causing reduced blood flow and metabolic depression[10]. This phenomenon is thought to be caused by interruption of the cortico-ponto-cerebellar pathways[18-20]; these pathways are formed by fibers that derived from areas of the cerebral cortex, project to the ipsilateral pontine nuclei, and send axons to the contralateral cerebellar cortex[10-11]. Focal cerebral injury causes axonal rupture or damage, which can disrupt neural signal transfer[21]. A decrease or absence of afferent input[8] can cause functional inhibition in the contralateral cerebellar hemisphere[22].

Decreased blood flow in the cerebellar hemisphere contralateral to a supratentorial stroke has been reported by a number of positron-emission tomography and single-photon emission CT studies[9-10, 14-15]. Ito *et al* [9] used positron-emission tomography to evaluate regional cerebellar blood flow in 20 patients with cerebrovascular disease, reporting cerebral hypoperfusion in the contralateral cerebellar hemisphere. Komaba *et al* [10] used single-photon emission CT to evaluate regional cerebellar blood flow in 113 patients, and confirmed that blood flow was reduced in the cerebellar hemisphere contralateral to a supratentorial stroke. DSC-PWI is an effective non-invasive technique for evaluating regional brain tissue perfusion to detect microcirculation[23-27], especially in acute and subacute stroke[28-29]. A small number of studies have examined cerebral hypoperfusion in the contralateral cerebellar hemisphere after subacute stroke using PWI[8, 11].

A previous study[8] using DSC-PWI imaging in 301 patients with acute stroke reported that the time to peak was higher, but the contralateral cerebellar blood flow was reduced by (22.75 ± 10.94%) compared with the ipsilateral cerebellum. However, Yamada *et al* [11] reported that contralateral cerebellar cerebral blood volume was reduced, while there was no significant change in cerebral blood flow and time to peak between the right and left cerebellar hemispheres. The current results showed clear differences in perfusion between cerebellar hemispheres in 10 patients with hyperacute unilateral supratentorial stroke. rNEI values were significantly different between the unaffected cerebellar hemisphere and the affected hemisphere. There was no significant difference in rMTE values or rNEI values between the hemispheres ($P > 0.05$, paired *t*-test). There are several possible explanations for these findings. First, the patients in our study were in the hyperacute stages of stroke within 6 hours of symptom onset, whereas the patients in previous studies were in the acute stages[8] or the subacute to chronic stages of stroke (range, 6–120 days)[11]. The different stages of stroke are associated with differences in perfusion, which may be reflected in the measurement of perfusion with DSC-PWI. Second, the MR imaging scanner used in our study was different from that used in previous studies; we used a 3.0T scanner, while previous studies used 1.0T[11] or 1.5T[8] scanners. This may have caused differences in the image resolution ratio.

The current results demonstrated abnormal contralateral cerebellar perfusion in six of 10 patients following unilateral supratentorial stroke, using 3.0 T DSC-PWI. The rNEI maps may have reflected brain ischemia. We found that rNEI values were lower in the contralateral cerebellar hemisphere compared to the ipsilateral hemisphere, consistent with a previous study[9]. In contrast, there was no significant difference in rMTE or rTTM values between the right and left cerebellar hemispheres. The stage of hyperacute stroke may cause a decline of regional neural activity, coupled with a change in cerebral blood volume and blood flow. Vascular responses were almost identical between the affected and unaffected hemispheres of the cerebellum, supporting the notion that the decrease in metabolism on the affected side was caused by vasoconstriction[30]. Therefore, the abnormalities of rMTE value and rTTM value in the contralateral cerebellum were likely to have been caused by vasoconstriction rather than a change of vascular blood velocity[30]. It remains unclear whether the infarct area in the supratentorial brain is correlated with the occurrence of diaschisis[8, 18]. The present results revealed four cases (40%) with a large infarction area and six cases (60%) with a smaller infarction area, all of which experienced abnormal cerebral hypoperfusion. These results indicate that the infarction area has no direct relationship to cerebral infarction area.

In conclusion, abnormal contralateral cerebellar hemisphere perfusion in patients with supratentorial stroke was successfully detected with DSC-PWI. DSC-PWI may thus provide a suitable tool for the study of cerebral hypoperfusion in the contralateral cerebellar hemisphere after stroke.

SUBJECTS AND METHODS

Design

A retrospective review in radiology.

Time and setting

Experiments were performed at the First Affiliated Hospital of Wenzhou Medical College, China from December 2007 to September 2010.

Subjects

A total of 17 patients with subacute stroke (≤ 6 hours) who presented at the First Affiliated Hospital of Wenzhou Medical College from December 2007 to September 2010 were included in this study.

Inclusion criteria of crossed cerebellar diaschisis: (1) patients exhibited symptoms of a focal neurological deficit; (2) patients were not suitable for CT examination due to iodine allergies or other reasons; (3) patients were evaluated with MRI within 6 hours of symptom onset, with abnormal DWI showing a unilateral supratentorial lesion; and (4) patients exhibited a decrease in DSC-PWI of the contralateral cerebellar hemisphere.

Exclusion criteria were as follows: (1) cerebral hemorrhage, brain tumor and other neurological diseases; (2) non-unilateral supratentorial stroke; (3) magnetic resonance angiography imaging showed abnormal vertebral basilar artery; (4) DWI and conventional T2-weighted MR imaging displayed pathophysiological changes in the cerebellum.

Among the 17 patients initially examined, 10 patients fulfilled the inclusion criteria. Of these 10 patients, eight were male and two were female, with ages ranging from 43 to 73 years, and a mean age of 60 years. Informed consent was given by participants and family members, and the study was conducted in accord with the Declaration of Helsinki.

Methods

Image acquisition and processing

All studies were performed using a 3.0T GE (GE, Medical System, USA) scanner, using the standard quadrature transmit-receive head coil. In addition to conventional T1-weighted (repetition time, 9 000 ms; echo time, 150 ms; flip angle, 90°; section thickness, 5 mm; matrix, 288 × 192; field of view, 24 × 24 cm) and T2-weighted (repetition time, 9 000 ms; echo time, 150 ms; flip angle, 90°; section thickness, 5 mm; matrix, 288 × 192; field of view, 24 × 24 cm), single-shot echo-planar imaging-spin echo diffusion-weighted MRI[31] were obtained (b_{max} = 1 000 s/mm^2[32-35]; repetition time, 5 300 ms; echo time, 62.6 ms; section thickness, 5 mm; matrix, 160 × 160; field of view, 24 × 24 cm), and echo-planar PWI images[35] were recorded during bolus injection of 0.2 mmol/kg of gadopentate dimeglumine (Gd-DTPA) at 5 mL/s using single shot gradient-echo echo-planar imaging (repetition time, 1 500 ms; echo time, 75 ms; flip angle, 90°; section thickness, 5 mm; matrix, 128 × 128; field of view, 24 × 24 cm). PWI scans were post-processed to generate maps of NEI and MTE, and three-dimensional fast imaging employing steady state acquisition with cycled phases magnetic resonance angiography (repetition time, 30 ms; echo time, 4.6 ms; flip angle, 15°; section thickness, 1.2 mm; matrix, 256 × 256; field of view, 18 × 18 cm).

We analyzed the perfusion abnormalities of the rNEI, rMTE and rTTM maps[36] from the original data of perfusion images analyzed with ADW 4.3 workstations using GE company special perfusion software processing for all sections that displayed a signal intensity abnormality by manual segmentation[37]. The regions of interest were approximately 150 pixels.

Image analysis

Anomalous signals in both sides of the cerebellum: mild, magnetic resonance perfusion figure presented no perfusion defect; moderate, magnetic resonance perfusion with mild perfusion defect; mild, obvious, magnetic resonance perfusion figure exhibited obvious perfusion defect. The data were evaluated by two experienced radiologists. In accord with the results, we analyzed the scores of the rNEI, rMTE and rTTM maps, and calculated the percentage of abnormal perfusion.

Statistical analysis

All data were analyzed using SPSS 16.0 software (SPSS, Chicago, IL, USA). Data in the rNEI, rMTE and rNEI were presented as mean ± SD. Means of variables were compared by a two-tailed *t*-test assuming unequal variances. A value of $P < 0.05$ was considered statistically significant.

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Author contributions: Pan Liang, Yuxia Duan and Hongqing Wang provided study data and ensured the integrity of the data. Weijian Chen and Yunjun Yang participated in study concept and design. Pan Liang and Yunjun Yang participated in data analysis. Pan Liang wrote the manuscript. Weijian Chen, Yunjun Yang and Xiaotong Wang were in charge of manuscript authorization, provided technical or material support, obtained the funding and served as principle investigator. Pan Liang, Yunjun Yang, Yuxia Duan and Hongqing Wang participated in statistical analysis.

Conflicts of interest: None declared.

Ethical approval: This study was approved by the Ethics Committee, First Affiliated Hospital of Wenzhou Medical College, China.

Supplementary information: Supplementary data associated with this article can be found, in the online version, by visiting www.nrronline.org, and entering Vol. 7, No. 12, 2012 item after selecting the “NRR Current Issue” button on the page.

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