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

Pan Liang¹, Yunjun Yang¹, Weijian Chen¹, Yuxia Duan¹, Hongqing Wang¹, Xiaotong Wang²

¹Department of Radiology, First Affiliated Hospital of Wenzhou Medical College, Wenzhou 325000, Zhejiang Province, China
²Second Affiliated Hospital of Wenzhou Medical College, Wenzhou 325000, Zhejiang Province, China

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

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 infarct[9-11]. Cerebral hypoperfusion in the contralateral cerebellar hemisphere after subacute stroke has been reported in perfusion abnormalities of the cerebellum after supratentorial unilateral hyperacute cerebral infarction[12]. 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 been reported by a number of single-photon emission computed tomography (SPECT) and positron-emission tomography (PET) studies[8-10, 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[6, 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).

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 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.

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    |

\[ t = 2.571, P = 0.037, \text{bold } 0.564 \]

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.

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). DSC (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.
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\[8, 9, 10, 14-15\]. Ito et al\[9\] used positron-emission tomography to evaluate regional cerebral blood flow in 20 patients with cerebrovascular disease, reporting cerebral hypoperfusion in the contralateral cerebellar hemisphere. Komaba et al\[11\] 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\[28\]. It remains unclear whether the infarct area in the supratentorial brain is correlated with the occurrence of diachisis\[8, 18\]. 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[28] were obtained (b_max = 1 000 s/mm^2) ; 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[30] 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 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.

Acknowledgments: We thank postgraduates Nan Wu, Qiuyun Tong and Yi Lin from Wenzhou Medical College for data analysis and case scanning.

Funding: This study was supported by the National Key Project of Scientific and Technical Supporting Program funded by the Ministry of Science and Technology of China during the 11th Five-Year Plan, No. 2007BA05B07; the Zhejiang Health Science and Technology Plan Project, No. 2011KYB050; the Wenzhou Science and Technology Plan Project, No. Y2004A014; the Wenzhou Significant Scientific Research Project, No. Y20070038.

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.

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.

REFERENCES

[1] Tsang A, Stobbe RW, Asdaghi N, et al. Relationship between sodium intensity and perfusion deficits in acute ischemic stroke. J Magn Reson Imaging. 2011;33(1):41-47.
[2] Mui K, Yoo AJ, Verduzco L, et al. Cerebral blood flow thresholds for tissue infarction in patients with acute ischemic stroke treated with intra-arterial revascularization therapy depend on timing of reperfusion. AJNR Am J Neuroradiol. 2011;32(5):846-851.
[3] Ma HK, Zavala JA, Churiol L, et al. The hidden mismatch: an explanation for infarct growth without perfusion-weighted imaging/diffusion-weighted imaging mismatch in patients with acute ischemic stroke. Stroke. 2011;42(3):662-668.
[4] Sobrado M, Delgado M, Fernández-Valle E, et al. Longitudinal studies of ischemic penumbra by using 18F-FDG PET and MRI techniques in permanent and transient focal cerebral ischemia in rats. Neuroimage. 2011;57(1):45-54.
[5] Ogata T, Nagakane Y, Christensen S, et al. A topographic study of the evolution of the MR DWI/PWI mismatch pattern and its clinical impact: a study by the EPITHET and DEFUSE Investigators. Stroke. 2011;42(6):1596-1601.

[6] Seitz RJ, Oberstrass H, Ringelstein AE, et al. Failed recovery from thrombolysis is predicted by the initial diffusion weighted imaging lesion. Cerebrovasc Dis. 2011;31(6):580-587.

[7] Zhou Q, Dong Y, Huang L, et al. Study of cerebrovascular reserve capacity by magnetic resonance perfusion weighted imaging and photoacoustic imaging. Magn Reson Imaging. 2009;27(2):155-162.

[8] Lin DD, Kleinman JT, Wityk RJ, et al. Crossed cerebellar diaschisis in subarachnoid hemorrhage: a new role for MR perfusion imaging. AJNR Am J Neuroradiol. 2009;30(4):710-715.

[9] Ito H, Kanno I, Shimosegawa E, et al. Hemodynamic changes during neural deactivation in human brain: a positron emission tomography study of crossed cerebellar diaschisis. Ann Neurol. 2002;51(4):249-254.

[10] Komada Y, Mishina M, Utsumi K, et al. Crossed cerebellar diaschisis in patients with cortical infarction: logistic regression analysis to control for confounding effects. Stroke. 2004;35(2):472-476.

[11] Yamada H, Koshimoto Y, Sadozo N, et al. Crossed cerebellar diaschisis: assessment with dynamic susceptibility contrast MR imaging. Radiology. 1999;210(2):558-562.

[12] Gharabawie OA, Whislaw IC. Parallel stages of learning and recovery after skilled reaching after cortical stroke: "oppositions" organize normal and compensatory movements. Behav Brain Res. 2006;175(2):249-262.

[13] Takasawa M, Watanabe M, Yamamoto S, et al. Prognostic value of subacute crossed cerebellar diaschisis: single-photon emission CT study in patients with middle cerebral artery territory infarct. AJNR Am J Neuroradiol. 2002;23(2):189-193.

[14] Dodick DW, Roarke MC. Crossed cerebellar diaschisis during migraine with prolonged aura: a possible mechanism for cerebellar infarctions. Cephalalgia. 2008;28(11):83-86.

[15] Agrawal KL, Mittal BR, Bhattacharya A, et al. Crossed cerebellar diaschisis on F-18 FDG PET/CT. Indian J Nucl Med. 2011;26(2):102-103.

[16] Salem A, Salem AF, Al-Ibraheem A, et al. Evidence for the use of PET for radiation therapy planning in patients with cervical cancer: a systematic review. Hematol Oncol Stem Cell Ther. 2011;4(4):173-181.

[17] Mak D, Corry J, Lau E, et al. Role of FDG-PET/CT in staging and follow-up of head and neck squamous cell carcinoma. J Nucl Med Mol Imaging. 2011;55(5):487-499.

[18] Wu J, Tarabishy B, Hu J, et al. Cortical calcification in Sturge-Weber Syndrome on MRI-SWI: relation to brain perfusion status and seizure severity. J Magn Reson Imaging. 2011;34(4):791-798.

[19] He D, Zhao L, Li L, et al. Changes of blood flow perfusion by MR and NF-kappabeta expression in the region of perihematoma after experimental intracerebral hemorrhage: a correlation study. Int J Neurosci. 2009;119(6):806-814.

[20] Miyashita M, Olov JM, Tong DC, et al. Yield of combined perfusion and diffusion MR imaging in hemispheric TIA. Neurology. 2009;72(13):1127-1133.

[21] Buerke B, Wittkamp G, Dziewas R, et al. Perfusion-weighted map and perfused blood volume in comparison with CT angiography source imaging in acute ischemic stroke different sides of the same coin? Acad Radiol. 2011;18(3):347-352.

[22] Wang YJ, Yao QL, Fang F, et al. A study of ischemic penumbra by perfusion and diffusion-weighted imaging at high-field MRI (7.0 T). Zhonghua Yi Xue Za Zhi. 2010;90(25):1773-1777.

[23] Yamauchi H, Okazawa H, Sugimoto K, et al. The effect of deafferentation on cerebral blood flow response to acetazolamide. AJNR Am J Neuroradiol. 2004;25(1):92-96.

[24] Li T, Gao PY, Hu QM, et al. Prediction of infarct core and salvageable ischemic tissue volumes by analyzing apparent diffusion coefficient without intravenous contrast material. Acad Radiol. 2010;17(12):1506-1517.

[25] Rehl L, Nellesmann HM, Ladekari M, et al. Ovarian metastases from ventilicular cancer diagnosed using diffusion-weighted 3T magnetic resonance imaging. Ugelerg Laeger. 2011;173(16-17):1212-1213.

[26] Liu X, Zhou L, Peng W, et al. Effect of intravenous gadolinium-DTPA on diffusion-weighted imaging for prostate lesions and normal tissue at 3.0-Tesla magnetic resonance imaging. Acta Radiol. 2011;52(5):575-580.

[27] Lin YC, Lin G, Chen YR, et al. Role of magnetic resonance angiography (MRA) status become a foremost factor in selecting optimal acute stroke patients for recombinant tissue plasminogen activator (rt-PA) thrombolysis beyond 3 hours? Neuror Res. 2009;31(4):355-361.

[28] Shah MK, Shin W, Parikh VS, et al. Quantitative cerebral MR perfusion imaging: preliminary results in stroke. J Magn Reson Imaging. 2010;32(4):796-802.

[29] Sobesky J, Thiel A, Ghaemi M, et al. Crossed cerebellar diaschisis in acute human stroke: a PET study of serial changes and response to supratentorial reperfusion. J Cereb Blood Flow Metab. 2005;25(12):1685-1691.

[30] Kajimoto K, Oku N, Kimura Y, et al. Crossed cerebellar diaschisis: a positron emission tomography study with L-[methyl-11C] methionine and 2-deoxy-2-[(18F)fluoro-D-glucose. Ann Nucl Med. 2007;21(2):109-113.

[31] Kim SE, Lee MC. Cerebellar vasoreactivity in stroke patients with crossed cerebellar diaschisis assessed by acetazolamide and 99mTc-HMPAO SPECT. J Nucl Med. 2000;41(3):416-420.

[32] Liu Y, Karonen JO, Nuutilen J, et al. Crossed cerebellar diaschisis in acute ischemic stroke: a study with serial SPECT and MRI. J Cereb Blood Flow Metab. 2007;27(10):1724-1732.

[33] Li T, Kolar B, Tian W, et al. MR perfusion-weighted imaging may help in differentiating between nonenhancing gliomas and nonneoplastic lesions in the cervicomedullary junction. J Magn Reson Imaging. 2011;34(1):196-202.

[34] Vatter H, Güresir E, Berkefeld J, et al. Perfusion-diffusion mismatch in MRI to indicate endovascular treatment of cerebral artery occlusion: a systematic review. Hematol Oncol Stem Cell Ther. 2011;4(4):371-376.