

Spontaneous reperfusion after stroke, beneficial or harmful? A study with proton MR spectroscopy and gadolinium enhancement on T1WI

C-S. Lee¹, C-Y. Chen¹, C-Y. Wang^{1,2}

¹Department of Radiology, Tri-Service General Hospital, Taipei, Taiwan, ²Electrical Engineering, National Taiwan University, Taipei, Taiwan

Background

Clinical studies have shown that about 30% of the initial middle cerebral artery (MCA) occlusions have spontaneous reperfusion by 24 hours and about 70% 1 week later (1). However, it is still unclear whether the restoration of blood flow plays a harmful, or, conversely, a beneficial specific effect with respect to the reperfused tissue. It is known reperfusion can cause the overproduction of oxygen radicals that exceed the ability of antioxidant cellular defenses to eliminate them. Radicals can change the blood-brain-barrier (BBB) permeability that is likely to induce ischemic edema and further exacerbate the development of injury (2). Therefore, BBB breakdown represents an acute mechanism whereby reperfusion may injure the brain. Based on these findings, we hypothesize that survived neuron capacity which can consume oxygen and reduce the level of oxygen radicals may be a major factor of affecting the reperfusion outcome. To test the hypothesis, we use MR method to investigate the correlation between NAA concentration (as a neuron marker) when reperfusion commenced and the maximum degree of BBB disruption that was indicated with post contrast T1 enhancement at chronic stage with luxury reperfusion.

Material and methods

A total of 17 ischemic stroke patients were performed with 3-4 times MR imaging from acute to chronic stages. Each patient underwent dynamic susceptibility-contrast (DSC) perfusion-weighted imaging and gadopentetate-enhanced T1-weighted imaging and MRS scanning. All MR images were acquired on a Siemens 1.5T Magnetom Vision Plus system (Erlangen, Germany). Reperfusion was confirmed by rCBV map derived from DSC imaging. A region of interest (ROI) was drawn manually to include the infarct region to represent the ischemic lesion. The degree of enhancement on T1W images was expressed as rT1 which is defined as ratio of signal intensity within an ROI to that of the contralateral homologous brain of similar area. rNAA values, which is defined as similar as rT1, were measured when reperfusion commenced. Correlation between rNAA and maximum rT1 was calculated.

Results

The strongest enhancements on T1W images occurred at chronic stage for all patients with rT1 ranged from 1.97 to 1.07 (mean rT1=1.62±0.30). 3 patients presented reperfusion at acute stage (< 2 days), 9 patients at subacute stage (< 9 days) and 4 patients at chronic stage (>13 days). Residue NAA at the time when reperfusion presented with rNAA ranged from 0.88 to 0.15 (mean rNAA=0.44±0.25). rT1 and rNAA were closely related with $r = -0.75$ (Figure 1).

Discussion

Our data demonstrated that survived neuron number at the time when reperfusion occurring after ischemia may be an affecting factor for the reperfusion to be beneficial or harmful. In condition of reperfusion with enough neuron capacity, injury of oxygen radicals may be smoothed. In contrast, if neuron capacity was not enough to consume the flood of radicals, reperfusion injury may be resulted. Abundant neuron capacity may also slow down the development of reperfusion injury, which may be a common factor for the diverse result of ischemic studies in human or animal with MR method such as the time course of ADC (3-5). However, the complex outcome of reperfusion must take into account the balance result between reperfusion blood volume and survived neuron number. Blood flow below or over the neuron demanded will cause different appearance of ischemic evolution.

References

1. Fieschi C, Argentino C, Lenzi GL, et al. *J Neurol Sci* 1989;91:311-321
2. Nelson CW, Wei EP, Povlishock JT, et al. *Am J Physiol.* 1992;263:H1356-H1362.
3. Huang IJ, Chen CY, Chung HW, et al. *Radiology* 2001;221:35-42.
4. William AC, Lee HS, et al. *Radiology* 2001;221:27-34.
5. Naoyuki M, Tsukasa N, Toshihiko K, et al. *AJNR Am J Neuroradiol* 2000;21:60-66.

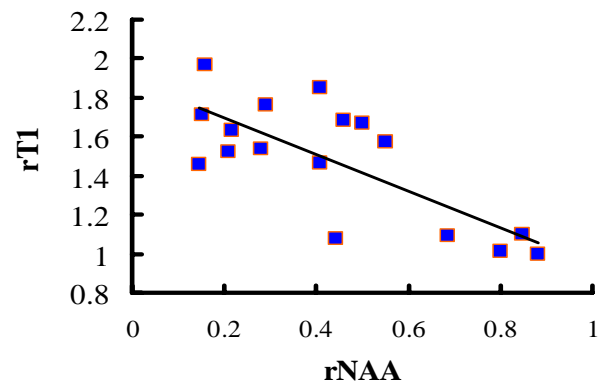


Fig. 1 Plot of the correlation between rT1 and rNAA. A negative correlation with $r = -0.75$ was found. The regression line is $y = -0.94x + 1.89$.