Monitoring pH and energy metabolim in subacute stroke using ^{31}P and ^{1}H MRSI

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Purpose

In vivo changes in tissue pH and energy metabolism are key to understanding stroke pathophysiology. Our goal was to study pH changes in subacute ischemic stroke and their relation to energy metabolism, which are, unlike acidosis in acute stroke, not yet well elucidated.

Methods

^{31}P MRSI was used to measured tissue pH and cell energy markers including ATP and phosphocreatine (PCr) in subacute stroke. Data were combined with ^{1}H MRSI providing the total creatine concentration as well as the neuronal marker N-acetylaspartate. Tissue pH was determined by the chemical shift difference between inorganic phosphate (Pi) and PCr. [1] 31P data were analysed with jMRUI. For stroke areas the Pi region in the spectrum was fitted using two Lorentzian signals (Figure, upper panel), with one fixed at 4.82 ppm representing healthy tissue. We included 19 patients with first-ever ischemic stroke (mean time after stroke: 6 days). In addition to pH analysis, metabolite concentrations were compared between ischemic tissue and contralateral (healthy) tissue using multivariate ANOVA with repeated measurements.

Results

In subacute stroke the Pi signal can be analysed with two peaks, one indicating a more alkalotic tissue fraction (pH 7.37±0.28 vs. 7.03±0.02 for healthy tissue). While the intensity of the alkalotic Pi signal increases with increasing stroke volume (Figure, middle panel), the respective pH-values stay constant (Figure, bottom panel). Furthermore, only a moderate decrease of energy-rich metabolites (phosphocreatine reduced by 17%, ATP reduced by 19%) was observed while total creatine was reduced by 51%.

Discussion

The finding of a tissue fraction with alkalotic pH in subacute ischemia confirms previous work by Levine et al. [2], who found a low field shift of the Pi signal in stroke tissue. In contrast to this previous study, which was performed at 1.5 T, we could identify two regions with different pH and only incomplete energy loss suggesting two differently viable cellular moieties, best explained by active compensatory mechanisms following acute cerebral ischemia.

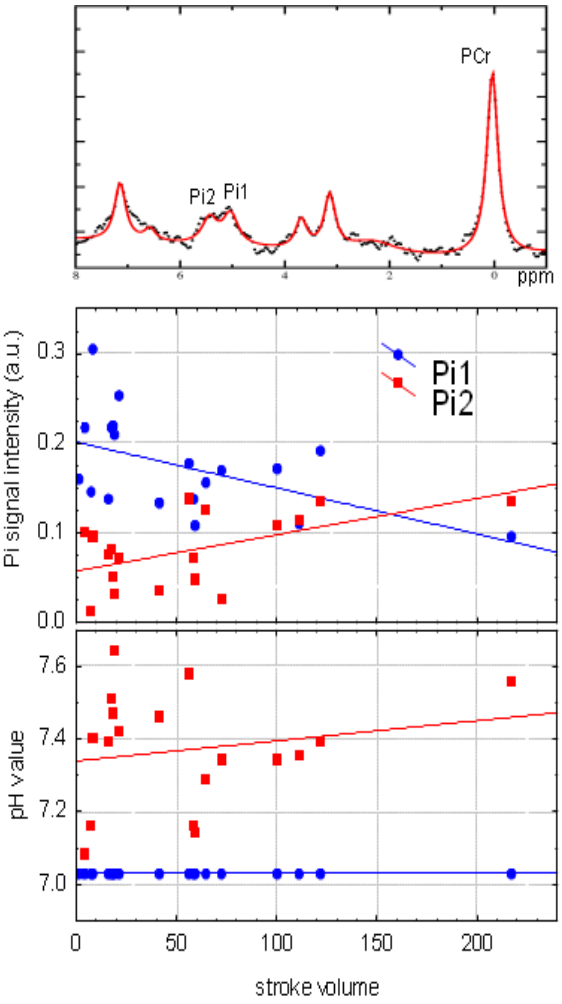

Figure: Upper panel: Low field region of the 31P spectrum showing the PCr and the two Pi signals. Middle panel shows signal intensity for Pi1 (blue) and Pi2 (red) as well as the respective pH (lower panel) as function of stroke volume.

References

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