N-Acetylaspartic acid

N-Acetylaspartic acid, or N-acetylaspartate (NAA), is a derivative of aspartic acid with a formula of C6H9NO5 and a molecular weight of 175.139.

NAA is the second-most-concentrated molecule in the brain after the amino acid glutamate. It is detected in the adult brain in neurons, oligodendrocytes and myelin and is synthesized in the mitochondria from the amino acid aspartic acid and acetyl-coenzyme A.

A neuronal marker. Normally the tallest peak (higher than Cr or Cho). ↓ in ≈ all focal and regional brain abnormalities (tumor, MS, epilepsy, Alzheimer's disease, abscess, brain injury…)

NAA is unique not only by virtue of its exceptionally high concentration in the brain, but also due to the powerful signal that it gives off in water-suppressed proton MRS spectrograms.

Glutamate was not associated with a high frequency of preoperative seizures in patients with gliomas. Instead, increased total N-Acetylaspartic acid (tNAA) was significantly associated with them. Moreover, the multivariable analysis indicated that an increased level of tNAA was an independent predictor of preoperative seizures. According to MRS analysis, tNAA, rather than glutamate, might be useful to detect preoperative seizures in a patient with supratentorial gliomas [1)].

Goncalves et al., hypothesized that changes in the concentration of N-acetylaspartate (NAA) in the motor and sensory cortices in the brain would emulate the time course of neurological recovery following decompression surgery for cervical spondylotic myelopathy (CSM). Their aim was to compare and contrast how metabolite levels in the motor and sensory cortices change after surgery to reverse downstream spinal cord compression.

Twenty-four patients with CSM and 8 control subjects were studied using proton magnetic resonance spectroscopic imaging (Proton magnetic resonance spectroscopic imaging) acquired on a 3.0-T Siemens MRI unit. The Proton magnetic resonance spectroscopic imaging data (TE 135 msec, TR 2000 msec) were acquired to measure absolute levels of NAA from the motor and sensory cortices in the cerebral hemisphere contralateral to the side of greater deficit at baseline in each subject. Data were also acquired at 6 weeks and 6 months following surgery. Control subjects were also evaluated at 6 weeks and 6 months following baseline data acquisition. Neurological function was measured in each subject at all time points using the Neck Disability Index (NDI), Modified Japanese Orthopaedic Association scale (mJOA), and the ASIA impairment scale.

In the motor cortex of patients, NAA levels decreased significantly ($p < 0.05$) at 6 weeks and 6 months postsurgery compared with baseline levels. In the sensory cortex of patients, NAA levels decreased significantly ($p < 0.05$) only at 6 months after surgery compared with baseline and 6-week levels. No significant changes in NAA were found in control subjects. Clinical scores demonstrated significant ($p < 0.05$) motor recovery by 6 weeks, whereas sensory improvements ($p < 0.05$)

appeared at only 6 months.

Findings suggest that metabolite changes in both the motor and sensory cortices mimic the time course of functional motor and sensory recovery in patients with CSM. The temporal course of neurological recovery may be influenced by metabolic changes in respective cortical regions [2].

1)
Nakae S, Kumon M, Murayama K, Ohba S, Sasaki H, Inamasu J, Kuwahara K, Yamada S, Abe M, Hirose Y. Association of preoperative seizures with tumor metabolites quantified by magnetic resonance spectroscopy in gliomas. Sci Rep. 2021 Apr 12;11(1):7927. doi: 10.1038/s41598-021-86487-6. PMID: 33846339.
2)
Goncalves S, Stevens TK, Doyle-Pettypiece P, Bartha R, Duggal N. N-acetylaspartate in the motor and sensory cortices following functional recovery after surgery for cervical spondylotic myelopathy. J Neurosurg Spine. 2016 Oct;25(4):436-443. PubMed PMID: 27176111.