1H-MRS study of obstructive sleep apnea syndrome

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Introduction.

Obstructive sleep apnea syndrome (OSAS)[1] is a common sleep disturbance affecting from 4 to 9% of the adult population, according to epidemiological findings. This syndrome is characterized by repeated breath arrests occurring during sleep, leading to repeated arterial oxygen desaturation. It is associated with sleep fragmentation and excessive daytime sleepiness and may result in mood changes and intellectual deterioration. We carried out 'in vivo' localized proton magnetic resonance spectroscopy on 20 patients to investigate the impact of OSAS on cerebral metabolite concentrations.

Subjects and methods.

Twenty patients with OSAS (4 females and 16 males, mean age: 52.8±11.9 years), participated in the study.
They underwent a complete physical examination, including neurological, cardiovascular, ear, nose and throat evaluations.
One-night polysomnographic study was performed according to the Guidelines of the American Electroencephalographic Society (1994)[2] . OSAS diagnosis was based on the Criteria of the International Classification of Sleep Disorders (1990).[3] The number of apnea or hypopnea events per hour was obtained by dividing the total number of such events per total sleep time as defined by the apnea-hypopnea index (AHI). In a separate session, proton MR spectra were acquired using 1.5 T MR-Imaging system (Signa LX-GE Medical Systems) with the standard head coil. VOIs for spectroscopy acquisitions, mainly white matter, were placed in frontal and temporal regions bilaterally. For VOI localization, FLAIR sequence was used, with TR/TE/TI 8000ms/120ms/2000ms, FOV=24x24 cm,slice thickness=5mm, 256x256 pixels. MRS acquisition was performed by a PRESS sequence with parameter: TR/TE 1500ms/40ms, 2048 points,128 averages, VOI size 4-6cc. FIDs were elaborated by: zero filling, lorentzian-gaussian apodization, FFT, baseline correction, gaussian fit for peak area calculation. Metabolite quantification was expressed as ratios with respect to the creatine peak area. ^{1}H MRS was also performed on 10 healthy age-matched control subjects with no systemic or neurological diseases. Analysis of variance (ANOVA) and Fisher's least significant difference (LSD) were used to compare the values of cerebral metabolites in the control group with those of the patient group.

Results.

Patients vs Controls	Temporal locations	Frontal locations
Ins/Cr	⇧ p<0.00001	⇧ p<0.04
Cho/Cr	⇧ p<0.00002	ns
NAA/Cr	ns	⇩ p<0.04

Significantly lower values of NAA/Cr ratio were disclosed in frontal regions of OSAS patients ($p<0.004$) compared with control subjects. A significant increase in Ins/Cr ratio was evident bilaterally in patient temporal and frontal regions ($p<0.00002$ and $p<0.04$). In the temporal regions of OSAS patients Cho/Cr ratio values were also significantly greater than those of controls. A significant correlation ($p<0.05$) was found between AHI index and NAA/Cr ratio values in the frontal regions of OSAS patients.

Discussion.

The reduction in NAA/Cr ratio in frontal regions in OSAS patients could be related to the neuronal dysfunction or neural loss in these areas. The increase in the Cho/Cr ratio in temporal regions and of Ins/Cr both in frontal and temporal regions could be interpreted as evidence for membrane breakdown and reactive gliosis, respectively. Metabolite changes could be a consequence of repeated episodes of hypoxia in OSAS patients even in the absence of detectable abnormalities at conventional MRI. A known mechanism of cerebral circulatory regulation increases cerebral blood flow in response to hypercapnia. Haemodynamic adjustment in the cerebral deep white matter may be not sufficient to compensate for a decrease in arterial oxyhaemoglobin saturation in OSAS patients. The resulting reiterate cerebral hypoxia insults due to repeated sleep apnea episodes can have as a consequence the metabolic impairment seen in the cerebral white matter.

References.

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