MR Proton Spectroscopy study in nocturnal frontal lobe epilepsy
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Target audience
Neurologists and neuroradiologist interested in diagnostic work-up of patient with frontal nocturnal epilepsy and motor parasomnias.

Purpose
Nocturnal frontal lobe epilepsy (NFLE) is a syndromic entity that includes paroxysmal episodes with polymorphic semeiology, and variable intensity and duration appearing almost exclusively during sleep. The NFLE can present in family form with mendelian autosomal dominant inheritance (ADNFLE), but most cases remain cryptogenetic. Accepted criteria for the diagnosis of nocturnal frontal lobe seizures are lacking and even ictal scalp EEG recording could fail to disclose paroxysmal abnormalities. Whereas some ictal features seem to involve fronto-mesial structures other ictal manifestations, such as complex semi-purposeful, sometime bizarre, motor behaviour (kicking, pedaling, pelvis thrusting, body rocking, etc.) accompanied by intense autonomic activation are clinically hard to confine to a specific area.

The pathophysiology of seizures in NFLE is not yet fully understood. The purpose of this study was to evaluate using proton MR spectroscopy (1H-MRS) the possible involvement of the anterior cingulate cortex, which is considered to have inhibitory function on spontaneous motor activity and the thalamus, were the spellep K-complex arise from.

Materials and Methods
From July 2011 to June 2012, 20 patients (age: 36 ±10 years, 12 F) with clinical diagnosis of NFLE, without parasomnias, and 16 healthy controls (age: 32±7 years, 12 F) were evaluated with a standardized MRI protocol in a 1.5T magnet with a phased array 8 channels coil. Each subject underwent MR examination using a 1.5 T GE Signa scanner, following the same protocol including: T1-weighted volumetric imaging (FSPGR, TR/TE=123.5/2.2 ms; 1 mm isotropic resolution); diffusion tensor imaging (TR/TE= 104/82 ms, 32 acquisitions with non-collinear field gradients b-values=600 mm2/s-1, axial oblique FOV=39 cm; 128×128 in-plane resolution; 2.5 mm slices); single voxel proton MRS with PRESS sequence (TR/TE=12.3/5.2 ms; 1 mm isotropic resolution); diffusion tensor imaging (TR/TE=104/82 ms, 32 acquisitions with non-collinear field gradients b-values=600 mm2/s-1, axial oblique FOV=39 cm; 128×128 in-plane resolution; 2.5 mm slices); T2-weighted FLAIR (TR/TE=85/86 ms, TI=2000 ms, axial FOV= 24 cm, 256×256 in-plane resolution, 3 mm slices); T2-weighted FSE (TR/TE=5.6-5.8 ms depending on slice number/107 ms, coronal FOV= 21 cm, 286×256 in-plane resolution, 1 mm slice).

Group differences were calculated using the Student T-test. Correlations were performed between 1H-MRS and clinical parameters, using the Pearson test 1-tailed (statistical significance: p<0.05).

All participants provided written informed consent for the protocol study approved by the local Ethics Committee.

Results
The mean disease duration was 23±13 years, the mean age of patients at the onset of the disease was 11±7 years, the mean age at the diagnosis was 20±11 years.

Brain structural MR changes were not present on conventional images. In patients the cortical anterior circuluated NAA/Cr (1.23, mean) was lower in comparison to healthy controls (1.38, mean) (p<0.01). Cortical NAA/Cr values were negatively correlated with clinical severity of NFLE (beta=-0.473, p<0.05) (fig. 2). No differences were found in the thalamic NAA/Cr (1.36) ratio compared to healthy controls (1.29).

Discussion and Conclusion
This study show an involvement of the anterior cingulate cortex, more severe in NFLE patients with higher seizure frequency, in the pathophysiology of NFLE. This result is consistent with the most frequently involvement of anterior cingulate cortex at ictal onset detected in patients with frontal epilepsy who underwent intracerebral EEG recording. Our result, obtained with non-invasive technique, should be taken into consideration when is difficult the differential diagnosis between NFLE and non-epileptic motor phenomena arising from sleep (parasomnias), based on clinical and conventional EEG.

However the role of the involvement of fronto-mesial structures have to be elucidated combining multiple advanced neuroimaging modalities and including in the study patients with different parasomnias.

References