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Case Report

MRS findings in electrical status epilepticus in sleep: Report of two cases

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Abstract

Background: To evaluate the changes in brain metabolites by H1 magnetic resonance spectroscopy in two patients with electrical status epilepticus.

Case Description: Two boys (aged 6 and 7 years) with electrical status epilepticus in sleep have been evaluated. N-acetyl aspartate levels were slightly elevated, and showed no decline in the postictal period. Creatine and choline levels were similar to that in controls. No evidence of neuronal cell damage was seen.

Conclusion: Electrical status epilepticus is a balanced condition of hypermetabolism, when not accompanied with seizure.

Key Words: Magnetic resonance spectroscopy, seizure, status epilepticus

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INTRODUCTION

Electrical status epilectus in sleep (ESES) is an electroencephalographic (EEG) finding of significant epileptiform discharges during sleep. Patry *et al.* defined EEG criteria of RSRS as a spike/wave index of 85%–100% on three or more recordings over a 1-month period.[6] ESES shows a wide range of clinical features. Loss of language, hyperactivity, memory deficits, and aggressiveness are common complaints.[3] Seizures are the presenting symptom of only 80% of patients with electrical status epilepticus.[8] The effects of continuous epileptiform discharges on cerebral metabolism could be responsible for nonepileptic symptoms. We performed brain magnetic resonance imaging and H^1 magnetic resonance spectroscopy (MRS) in two patients with ESES.

CASE REPORT

Two boys, one aged 6 years old and the other 7 years, with atypical absence epilepsy showed continuous spike and wave pattern on their EEGs for at least 85% of their non-REM sleep. In repeated EEG recordings their electrical status epilepticus in sleep persisted for 2 and 3 months, respectively. The 6-year-old boy also had some difficulties in speech, without auditory agnosia. Both were on treatment with levetiracetam and clobazam. The control case was a 7-year-old boy who had a scheduled MRI for recurrent headaches. Magnetic resonance imaging of the two cases and the control was normal.

MR spectroscopy studies of the patients were performed both at awakening and 1 hour after the beginning of sleep. The MR spectroscopy voxels were placed into the left frontal lobe and the left thalamus [Figure 1]. Quantification of MR spectroscopy data was performed by the internal water standard method. We obtained water-suppressed and nonsuppressed MR spectra (STEAM; TR=3500 msec). The integral values of N-acetyl aspartate (NAA), creatine (Cr), choline (Cho), and tissue water peaks were calculated. Tissue water was

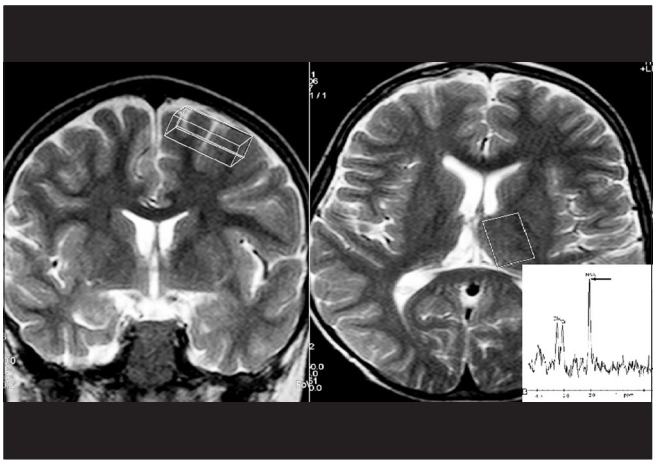

Figure 1: Slightly increased levels of N-acetyl aspartate in magnetic resonance spectroscopy of patient 1

used as a reference metabolite, and the integral values of the other neurometabolites were proportioned to the integral values of tissue water.

The levels of NAA was slightly higher in ESES patients compared to the control patient. The Cho and Cr values in the patients were nearly identical to that in the control [Table 1].

Table 1: The metabolite levels in the thalamic and frontal area

	Case 1		Case 2		Control	
	Awake	Sleep	Awake	Sleep	Awake	Sleep
Thalamus						
NAA/Water	0.37	0.35	0.39	0.40	0.29	0.27
Cr/Water	0.34	0.26	0.25	0.23	0.33	0.30
Cho/Water	0.22	0.21	0.19	0.20	0.17	0.14
Frontal cortex						
NAA/Water	0.46	0.37	0.39	0.39	0.35	0.33
Cr/Water	0.22	0.18	0.20	0.18	0.21	0.18
Cho/Water	0.13	0.14	0.11	0.11	0.13	0.13

NAA: N-acetyl aspartate, Cr: creatine, Cho: choline

DISCUSSION

During an epileptic seizure, neurons in the brain discharge repetitively in a hypersynchronous fashion. Prolonged seizures cause decrease in ATP and increase in AMP and ADP, as well as substances like lactate. Intracellular calcium influx activates phospholipases, leading to an increase in free fatty acids and prostaglandins and thus causing neuronal damage.[2,7]

MR spectroscopy provides a measure of brain metabolites. Each metabolite appears at a specific ppm, and each one reflects specific cellular and biochemical processes. NAA is a neuronal marker, and its level decreases with any disease that adversely affects neuronal/axonal integrity. Cr provides a measure of the energy stores. Cho is a measure of increased cellular turnover and is elevated in tumors and inflammatory demyelinating processes.[5] Previous human and animal studies on metabolic markers in status epilepticus have displayed increased NAA levels during seizure, followed by postictal decrease due to neuronal cell loss.[1,4] Higher NAA levels of patients at both the ictal (sleep) and postictal (awake) phases, indicates absence of neuronal cell loss in ESES. Increased NAA levels could be explained by a continuous hypermetabolic state of neurons. This has also been shown in an experimental animal study that choline levels increase during seizure.[9] Stability of the Cho levels in ESES despite the electrical activity of neurons has been considered as an indicator of cellular integrity. Lack of change in Cr levels was interpreted as showing steady energy balance during the increased electrical activity of neurons. Similar decline in Cr levels of three patients during sleep is connected to the physiologic changes of the sleep.

A limitation of this study is the paucity of patients, with only one control. However, demonstration of sustained neuronal integrity in the patients can be evaluated as an individual parameter.

In conclusion, prolonged seizure, a state of increased electrical activity and metabolism of neurons is known to lead to cellular damage. However, contrary to this, the preliminary data obtained from these two patients indicates that electrical status is a balanced condition of hypermetabolism, when not accompanied with seizure.

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