Serum Orexin-A Levels in Childhood Generalized Epilepsies

Gunce Basarir[1], Banu Basok[2], Nihal Olgac Dundar[3], Pinar Gencpinar[3]

[1] University of Health Sciences, Haseki Training and Research Hospital, Department of Pediatric Neurology, Istanbul, Turkiye
[2] University of Health Sciences, Behcet Uz Training and Research Hospital, Department of Medical Biochemistry, Izmir, Turkiye
[3] Izmir Katip Celebi University, Faculty of Medicine, Department of Pediatric Neurology, Izmir, Turkiye

INTRODUCTION

The process of neuroinflammation in the pathogenesis of epilepsy has been taken into consideration as an etiologic factor for decades. The role of immune system in the course of epilepsy has been first proposed in the 1960s.[4] Today, an increasing amount of evidence from both animal models and human studies indicates that brain inflammation is associated with epilepsy.[5-6]

Orexins are neuropeptides involved in the regulation of circadian rhythm, arousal, attention, modulation of emotions, appetite, cognition.[1] The orexin system mediates its activity via two endogenous ligands, orexin A and orexin B. Recent studies suggested that orexin A might have a neuroprotective and anti-neuroinflammatory function.[2-3] The involvement of this anti-inflammatory neuropeptide is one of the unclear mechanisms in the pathogenesis of epilepsy. Despite of supportive results of animal studies, there is lack of clinical data about the role of orexin-A in epilepsy.

OBJECTIVES

Considering the evidence for orexin A as a mediator that may be involved in the neuroprotection and anti-inflammatory processes in the brain, we hypothesized that an altered serum orexin-A level might be related to pathogenic mechanisms of generalized epilepsy.

To address this issue, we investigated serum orexin A concentrations of children with generalized epilepsy and control subjects. In addition, we evaluated the association between seizure semiology and frequency, and electroencephalographic findings and serum orexin A levels.

METHODS

Twenty-one children aged between 3-18 years who were followed-up with the diagnosis of generalized epilepsy and 21 healthy subjects as a control group were included to this cross-sectional case control study. A detailed neurological examination and detailed history were performed on all the patients. Serum orexin-A levels of the subjects were measured by enzyme-linked immunosorbent assay method.

RESULTS

No significant difference was found between serum orexin-A levels of the patients with generalized epilepsy and the control group (2745.33±2240.53 pg/ml, 2674.3±1794.5 pg/ml respectively; p=0.91). Moreover, There was no significant relationship between serum orexin-A levels and seizure frequencies, types, or electroencephalographic findings (all p>0.05).

CONCLUSIONS

This is the first clinical study investigating the role of orexin system in childhood generalized epilepsies and the possible relations between serum orexin-A levels and the patient characteristics affecting the course of disease.

Table I: Comparison of the demographic characteristics and serum orexin A levels

		Patient group (n=21)	Control group (n=21)	Total (n=42)	P value
Age (year)	Mean±SD	11.6±3.6	11.3±4.5	11.5±4	0.78
	Range	4.6-18.3	3.3-18	3.3-18.3	
M/F	M (%)	11 (42.4)	9 (42.9)	20 (47.6)	0.76
	F (%)	10 (47.6)	12 (57.1)	22 (52.4)	
Serum Orexin A (pg/ml)	Mean±SD	2745.3±2240.5	2674.3±1794.5	2709.8±2005.2	0.91
	Range	373.3-10587.9	310.6-5036.4	310.6-10587.9	

F, female; M, male; n, number; pg/ml, picogram/milliliter; SD, standard deviation.

The study showed that serum orexin-A levels did not significantly differ between children with generalized epilepsies and the healthy control group.

However, the link between the orexinergic system and epilepsy may be important for a better understanding of the etiopathogenesis of childhood generalized epilepsies. In the future, more clinical studies are needed to prove neuromodulatory role of this anti-inflammatory and neuroprotective peptide in the course of epilepsy.

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CONTACT

Gunce Basarir, MD
: guncebasarir@gmail.com.
: +905059356330