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Sleep Architecture in Patients with Juvenile Myoclonic Epilepsy
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Abstract:
The aim is to analyze the sleep architecture using polysomnography (PSG) in patients with Juvenile Myoclonic Epilepsy (JME): (newly diagnosed and those on valproate drug) attending epilepsy clinic at Alexandria University Hospitals.

Methods: This study involved 20 patients with JME on valproate (age: 22.40 ± 5.80 years; M: F = 6:14), 20 newly diagnosed patients (age: 18.55 ± 6.0 years; M: F = 6:14), and 20 matched healthy controls (age: 22.10 ± 5.0 years; M: F = 6:14). Clinical assessment, electroencephalogram (EEG), evaluation with comprehensive sleep questionnaire, and PSG were done for all patients.

Results: PSG showed significant alterations in sleep architecture in the total JME group in the form of reduced mean sleep efficiency (p = 0.001*), increased mean Rapid eye movement (REM) onset latency (p = 0.046*), decrease mean REM percentage (p = 0.011*), increased mean wakefulness after sleep onset (p = 0.018*), increase the index of total arousal (p = 0.005*), increased mean periodic limb movement index (P = 0.001*), and reduced apnea hypopnea index (P = <0.001) in comparison to control group. Valproate treated group showed increased sleep efficiency (p = 0.040*), decreased REM arousal index (P = 0.012), longer stage 3 (P = 0.038), and prolonged stage 2 (P = 0.049*) than the newly diagnosed group.

Conclusions: Sleep architecture was significantly disturbed in JME, with improvement in sleep efficiency in valproate treated patients.

Keywords: Juvenile myoclonic epilepsy, Valproic acid, Polysomnography, Sleep architecture.

Introduction: Juvenile Myoclonic Epilepsy syndrome is a good example for studying the relation between epilepsy and sleep through the circadian distribution of its seizures, precipitation by sleep deprivation and its different seizures types.(1) It also causes excessive day time sleepiness and also affects the quality of night sleep as well. (2) Moreover, it causes disturbances at the level of sleep microstructure in the form of reduced sleep efficiency, prolonged wakefulness duration and longer sleep onset latency. (3) Sleep disturbance leads to poor treatment compliance and poor seizures control. The abnormal arousal mechanism during sleep is an important key for sleep disturbance; it leads to activation of epileptiform activities. (4)(5) According to 1989 International Classification of Epilepsies
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