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EVALUATION OF AWAKE ELECTROENCEPHALOGRAPHY FINDINGS IN CHILDREN WITH ATTENTION DEFICIT HYPERACTIVITY DISORDER BEFORE PSYCHOSTIMULANT TREATMENT

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Attention deficit hyperactivity disorder is one of the most common neuropsychiatric disorders in childhood. Electroencephalographic abnormalities may be observed but it is controversial to perform electroencephalography before starting the treatment. Awake electroencephalography findings, demographic and psychometric variables of patients with attention deficit hyperactivity disorder treated between January 2005 and December 2010 were retrospectively evaluated. The study included 386 patients aged 4-18 (mean 9.61±3.04) years, who were diagnosed with attention deficit hyperactivity disorder according to the DSM-IV-TR (2000) diagnostic criteria. Epileptiform electroencephalography pattern was observed in 22/386 (5.7%) patients. Seven of 386 (1.8%) patients had a history of epilepsy diagnosis and antiepileptic medication. When patients with seizure history (with or without epilepsy diagnosis) and those with epileptic abnormalities on electroencephalography were excluded, the incidence of epileptiform abnormalities in attention deficit hyperactivity disorder patients was 12/386 (3.1%). After psychostimulant medication, epileptic seizures occurred in only three patients with epilepsy. The prevalence rate of epileptiform discharges on awake electroencephalography, observed in attention deficit hyperactivity disorder patients before psychostimulant treatment is similar to that in healthy schoolchildren. Since the seizures increased only in epileptic patients, we do not recommend routine awake electroencephalography evaluation in children with attention deficit hyperactivity disorder before psychostimulant medication.

Descriptors: ATTENTION DEFICIT DISORDER WITH HYPERACTIVITY; ELECTROENCEPHALOGRAPHY; CHILD, PRESCHOOL; ADOLESCENTS

INTRODUCTION

Attention deficit hyperactivity disorder (ADHD) is characterized by inattention, impulsivity and hyperactivity, which lead to functional disabilities. ADHD is a common disorder in childhood and affects 5%-7% of the general population (1). The Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV-TR (2000)) diagnosis requires the presence of six out of nine behavioral and functional symptoms of attention deficit or hyperactivity-impulsivity for a duration of at least six months, with onset before age seven years (2). ADHD occurs 5-9 times more often in boys as in girls.

Symptoms of ADHD are more common in patients with epilepsy than in general pediatric population (3). Several hypotheses have been proposed for the possible pathophysiology between ADHD and epilepsy including genetic predisposition, noradrenergic system dysregulation, chronic effects of seizures, effects of antiepileptic drugs and effects of stimulants (4, 5). Animal models of ADHD also suggest that synaptic abnormality in glutamatergic transmission may contribute to vulnerability for epilepsy and ADHD (6).

Many different abnormal electroencephalography (EEG) patterns can be found in patients with ADHD. These abnormalities include focal or generalized spikes, bilaterally synchronous spikes wave complexes and non-epileptiform abnormalities (7). The frequency of epileptiform discharges on EEG ranges from 5% to 60% without a history of seizures (8). The antiepileptic treatment may abolish epileptiform discharges and even improve ADHD symptoms (9). EEG studies of children with ADHD also revealed increased non-epileptiform theta activity primarily in the frontal regions, and increased delta, decreased alpha and beta activity in the posterior regions (10, 11).

It is controversial to perform an EEG in children with ADHD before starting the treatment (2). There is not enough evidence in the literature for performing EEG, but EEG monitoring can be useful to detect the unrecognized seizures. On the other hand, antiepileptic treatment of the children with ADHD who have epileptiform discharges without clinical seizures is also controversial.

Methylphenidate (MPH) and atomoxetine are the most effective drugs in reducing ADHD symptoms (12). MPH is a psychostimulant agent and common side

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effects include appetite loss, abdominal pain, insomnia and headache (13). Various formulations of MPH with different pharmacological profiles have been developed and they have been found effective in children with ADHD (14). In patients treated for ADHD, an increased risk of seizures is controversial. One study mentioned increased seizure risk with increasing dose of the stimulant compared with placebo in ADHD patients with comorbidity (15).

The aim of this study was to assess the prevalence of epileptiform abnormalities on awake EEG and the risk of seizure activity associated with psychostimulant treatment in patients with ADHD.

PATIENTS AND METHODS

Patients diagnosed with ADHD according to the DSM-IV-TR (2000) criteria between January 2005 and December 2010 were included in the study. The files of the patients were retrospectively evaluated in regard to gender, age, history of seizures, Wechsler Intelligence Scale for Children (WISC-R) results, type of treatment for ADHD and awake EEG pattern. Children with ADHD were referred for digitized routine EEGs, recorded for 20 minutes, including hyperventilation and photic stimulation with 20 electrodes and electrodes placed according to the international 10-20 system. No sleep tracings were recorded. All EEGs were performed in a single laboratory at Dokuz Eylul University Hospital, which is a tertiary pediatric referral center. The EEGs were recorded by using a Nihon-Kohden, Neurofax EEG-1000 version 05-73, with a sampling rate of 200 Hz. Generalized discharges of 3-Hz spike and slow waves, generalized discharges of spikes or multiple spikes, sharp slow wave complexes and focal spikes were accepted as epileptiform and EEGs with a disturbance in background cerebral activity (slowed rhythmicity or laterality of basic waves) without a seizure history were accepted as non-epileptiform EEG abnormalities. All EEGs were evaluated by a single child neurologist who was blinded to the clinical and diagnostic information of the patients. EEGs were first classified as either normal or abnormal. Secondly, abnormal EEGs were classified as epileptiform or non-epileptiform, and lastly the patients with epileptic abnormalities (EAs) were subdivided into those with focal, generalized and secondly generalized EAs. The study was approved by the local ethics committee of Dokuz Eylul University Medicine Faculty in Izmir.

Statistical methods

Data were analyzed with Statistical Package for the Social Sciences (SPSS), Version 15.0. Group differences were analyzed using the χ^2-test for categorical data and paired-sample Student's *t*-test for continuous variables. A p-value <0.05 was considered statistically significant.

RESULTS

We identified 386 patients aged 4-18 (mean 9.61±3.04) years meeting the DSM-IV-TR (2000) diagnostic criteria for ADHD. The study population included 84/386 (21.8%) female and 302/386 (78.2%) male patients. Out of 386 patients, 321 (83.2%) were treated with MPH and 65 (16.8%) were treated with other treatment strategies.

A previous history of seizures was recorded in 17/386 (4.4%) children; 7/17 (41.2%) patients had a history of epilepsy diagnosis and antiepileptic medication, 5/17 (29.4%) had nonepileptic seizures and 5/17 (29.4%) had the first epileptic seizure that was not treated. Of the patients with a diagnosis of epilepsy, 1/7 (14.3%) had absence epilepsy, 2/7 (28.6%) had frontal lobe epilepsy, 3/7 (42.8%) had rolandic epilepsy and 1/7 (14.3%) had occipital lobe epilepsy. Ten of 17 (58.8%) patients had epileptiform abnormalities on EEG before the treatment of ADHD and 5/10 (50%) of them had a previous diagnosis of epilepsy; 3/7 (42.8%) patients with a history of epilepsy diagnosis and antiepileptic medication developed epileptic seizures after MPH treatment. None of the patients with epileptiform discharges on awake EEG without a history of epilepsy diagnosis developed seizure after MPH treatment.

Abnormal awake EEG pattern (epileptiform or non-epileptiform) was observed in 56/386 (14.5%) children. Of these, 34/386 (8.8%) were non-epileptiform and 22/386 (5.7%) were epileptiform. Focal, generalized and secondly generalized EAs were detected in 11/22 (50%), 3/22 (13.6%) and 8/22 (36.4%) patients, respectively. Of the patients with focal EAs, 5/11 (45.4%) had centro-temporal spikes, 4/11 (36.4%) had frontal spikes and 2/11 had occipital sharp slow wave complexes (18.2%). All patients with focal EAs had normal cranial magnetic resonance imaging findings. The prevalence of epileptiform discharges on awake EEG in our study group was 22/386 (5.7%). When we excluded patients with seizure history (with or without epilepsy diagnosis) and those that had EAs on EEG, the incidence of EAs in ADHD patients was 12/386 (3.1%).

The WISC-R results were achieved from 40/56 patients with abnormal EEG pattern. The mean WISC-R score of patients with EAs on EEG was 94.5±13.20 and the mean WISC-R score of patients with non-epileptiform abnormalities on EEG was 88.17±22.67. There was no significant difference between the epileptiform and non-epileptiform ADHD groups according to WISC-R results (p>0.05). Psychometric variables of these 40 patients are summarized in Table 1.

Table 1. *Psychometric variables of patients with electroencephalographic abnormalities*

	Epileptiform ADHD group N = 16	Non-epileptiform ADHD group N = 24	p
Age (mean ± SD)	8±1.79	8.54±2.15	0.410
WISC-R verbal score (mean ± SD)	94.5±13.20	88.17±22.67	0.272
WISC-R performance score (mean ± SD)	101.69±13.95	94.17±22.58	0.243
WISC-R total score (mean ± SD)	98.56±13.24	91.33±22.47	0.254

ADHD = attention deficit hyperactivity disorder; WISC-R = Wechsler Intelligence Scale for Children

DISCUSSION

The reported rate of epileptiform abnormalities during awake EEG recordings observed in healthy schoolchildren was 3.5% (16). Although it is known that epileptiform activities are more frequent during sleep recordings, Capdevila et al. report the prevalence of epileptiform activity in healthy children during sleep to be 1.45% (17). On the other hand, the prevalence of epileptiform abnormalities

in ADHD children ranged from 5% to 60% in various studies (8, 16). These different prevalence rates possibly indicate methodological differences. Despite these increased prevalence rates, only a small percentage of ADHD children with EAs on EEG have epileptic seizures. Therefore, the clinical utility of routine EEG in children with ADHD appears to be limited (8). In previous studies, the prevalence of EAs in children with ADHD depended on the EEG according to sleep and/or awake monitoring. Millichap et al. demonstrated that the prevalence of EAs in non-epileptic children with ADHD was 26.1%, provided that sleep records were obtained (1). In wake recordings, the prevalence was only 7% (19). Hughes et al. report a prevalence of 30%, also mainly in sleep recordings (7). Sleep deprived EEG has an activating impact on epileptiform discharges (20). Socanski et al. also report the incidence of EAs in non-epileptiform ADHD children during awake recordings to be 5.4% (21). In the present study population, the prevalence of EAs on wake recordings was 5.7%. When patients with epileptic seizure history were excluded, the incidence of EAs during awake EEG recordings in ADHD patients was 3.1% and this epileptiform abnormality rate is similar to that in healthy children (16).

There is a complex relationship between epilepsy, ADHD and epileptiform activity. Epileptiform discharges are predominantly focal, mostly bilateral or right sided, distributed over rolandic areas in ADHD (22). Hughes et al. report the prevalence of epileptiform focal discharges during sleep recordings to be 24% (7). Silvestri et al. report on the focal, mainly centro-temporal spikes to be found in 51% of patients with ADHD (23). Similar to previous reports, EAs were mainly focal and secondly generalized (11/22 (50%), 8/56 (36.4%), respectively) in our study population. Of patients with focal EAs, 5/11 (45.4%) had centro-temporal spikes, 4/11 (36.4%) had frontal spikes and 2/11 had occipital sharp slow wave complexes (18.2%). All patients with focal EAs had normal cranial magnetic resonance imaging findings.

On the other hand, children with epilepsy have a significant risk of ADHD. Clinical studies suggest an ADHD prevalence of 30%-40% in patients with epilepsy (3, 24). The underlying mechanisms are still poorly understood. Frontal lobe epilepsies share behavioral features with ADHD, but in these patients seizure control does not guarantee concomitant improvement of the hyperactive behavior (25, 26). Nocturnal seizures significantly reduce sleep efficiency and increase drowsiness. Sleep disruption could be a cause of inattention and hyperactivity, so that it can be frequently associated with ADHD symptoms (27). ADHD is the most common psychiatric diagnosis in children affected by absence epilepsy (28). In addition to these, some antiepileptic medications have side effects, which include drowsiness, inattention, restlessness and decreased learning potential. Phenobarbital, benzodiazepine and topiramate are known to cause behavioral activity that may exacerbate underlying ADHD symptoms. Seven of our patients had a diagnosis of epilepsy and of them 1/7 (14.3%) had absence epilepsy, 2/7 (28.6%) had frontal lobe epilepsy, 3/7 (42.8%) had rolandic epilepsy and 1/7 (14.3%) had occipital lobe epilepsy. Five of them were treated with valproic acid and two with oxcarbazepine.

Clarke et al. report that low IQ children with ADHD have similar EEG abnormalities to those with normal IQ (29). On the other hand, Silvestri et al. describe a negative relation between the presence of nocturnal seizures and WISC-R IQ total and verbal scores (23). Similar to Clarke et al., we did not find any significant difference between the epileptiform and non-epileptiform ADHD groups according to WISC-R results ($p>0.05$).

Although case reports have warned about seizures in patients treated with MPH, clinical studies have noted significant improvements in ADHD symptoms without aggravation of seizures in patients with epilepsy (30). MPH affects the presynaptic reuptake of noradrenaline and dopamine but has no effect on neurotransmitters, which have been associated with the pathophysiology of epilepsy (2). Gross–Tsur et al. advise caution when using MPH in ADHD children with uncontrolled active seizures under treatment with antiepileptic drugs (31). If the patient has centro-temporal (rolandic) spikes and/or is treated with antiepileptic drugs, the treatment of ADHD with MPH is associated with a significant increase in clinical seizures (15, 32). Although current opinion is against performing EEG, the knowledge of an abnormal recording might influence the choice of medication to be used in the treatment of ADHD, non-stimulant *vs*. stimulant, or at least the formulation and dosage of medication considered safe. Bakke et al. recommend that, in patients with ADHD without seizures who do not respond to traditional ADHD drugs, an EEG with sleep recording should be performed and in the presence of EAs the use of an antiepileptic drug should be considered (33). In our study group, 3/7 (42.9%) patients developed epileptic seizures after MPH treatment and all of them had a history of epilepsy diagnosis, antiepileptic medication and had epileptiform activity before the treatment of ADHD with MPH. None of the patients had uncontrolled active seizures. Besides the psychostimulant medication, seizure aggravation may be the normal course of epileptic disease. None of the patients with epileptiform discharges without epilepsy developed seizure after MPH treatment.

In conclusion, before the treatment of ADHD with psychostimulant drugs, the awake and sleep EEG is indicated in any child with symptoms of ADHD and epilepsy. MPH treatment increased epileptic seizures only in patients with the diagnosis of epilepsy; therefore, MPH treatment should be used with caution in patients with a history of epilepsy diagnosis and treatment.

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S a ž e t a k

PROCJENA ELEKTROENCEFALOGRAFSKIH NALAZA U BUDNOM STANJU KOD DJECE S POREMEĆAJEM POZORNOSTI S HIPERAKTIVNOŠĆU PRIJE LIJEČENJA PSIHOSTIMULANSIMA

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Poremećaj pozornosti s hiperaktivnošću je jedan od najčešćih neuropsihijatrijskih poremećaja u djetinjstvu. Mogu se zapaziti elektroencefalografske nenormalnosti, ali je proturječno provoditi elektroencefalografiju prije početka liječenja. Retrospektivno su procijenjeni nalazi elektroencefalografije, demografske i psihometrijske varijable bolesnika s poremećajem pozornosti i hiperaktivnošću liječenih od siječnja 2005. do prosinca 2010. godine. U studiju je bilo uključeno 386 bolesnika u dobi od 4 do 18 (srednja dob 9,61±3,04) godine s dijagnozom poremećaja pozornosti s hiperaktivnošću prema dijagnostičkim kriterijima DSM-IV-TR (2000.). Epileptiformni elektroencefalografski nalaz zabilježen je kod 22/386 (5,7%) bolesnika. Sedam od 386 (1,8%) bolesnika imalo je u anamnezi dijagnozu epilepsije i terapiju antiepileticima. Kad su isključeni bolesnici s anamnezom konvulzija (s dijagnozom epilepsije ili bez nje) i oni s epileptičnim nenormalnostima na elektroencefalografiji, incidencija epileptiformnih nenormalnosti kod bolesnika s poremećajem pozornosti i hiperaktivnošću bila je 12/386 (3,1%). Nakon terapije psihostimulansima epileptične konvulzije nastupile su u samo troje bolesnika s epilepsijom. Učestalost epileptiformnih izbijanja tijekom elektroencefalografije u budnom stanju zabilježena u bolesnika s poremećajem pozornosti s hiperaktivnošću prije terapije psihostimulansima slična je onoj kod zdrave školske djece. Kako su se konvulzije povećale samo kod epileptičnih bolesnika, ne preporuča se rutinska elektroencefalografska procjena u budnom stanju kod djece s poremećajem pozornosti i hiperaktivnošću prije terapije psihostimulansima.

Deskriptori: POREMEĆAJ POZORNOSTI S HIPERAKTIVNOŠĆU; ELEKTROENCEFALOGRAFIJA; DJETE, PREDŠKOLSKO; ADOLESCENTI

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