

ELECTROENCEPHALOGRAM FINDINGS IN SAUDI CHILDREN WITH DELAYED LANGUAGE DEVELOPMENT

Taha Sadig Ahmed¹, Abdul Majeed Al Drees², Sultan Ayoub Meo³,
Gazza Ahmed⁴, Muhammad Al-Nasser M⁵

ABSTRACT

Objectives: To determine the possible relation between delayed language development (DLD), epilepsy and epileptiform Electroencephalogram (EEG) discharges in Saudi children.

Methodology: This study was conducted in King Abdulaziz University Hospital, King Saud University, Riyadh, Saudi Arabia. In this study 116 pediatric patients with delayed language development and similar number of age and sex matched controls were studied. The age range for both groups was 2-8 years. Children with intelligence quotient less than 70, hearing impairment and radiological evidence of structural cerebral disease were excluded from the study. Sleep and awake EEG was recorded in each case. The EEG was reviewed by a clinical neurophysiologist and a pediatric neurologist.

Results: Within the DLD group of children, 12.07% had epilepsy. This is significantly different ($p < 0.001$) from the control group in which none of the children had epilepsy. In DLD group 26 patients (22.41%) had epileptiform discharges in their Electroencephalogram. However, in control group only one child (0.86%) had epileptiform discharges. The delayed language development patients had a significantly higher percentage of abnormal EEGs, $p < 0.001$ compared to the control group. Furthermore, out of the 102 DLD patients, who had no seizure, 12 (11.76%), had epileptiform discharges in their EEG.

Conclusions: It is concluded that the children with delayed language development are associated with higher prevalence of epileptiform EEG discharges and epilepsy compared to their control.

KEY WORDS: Electroencephalogram, Delayed Language Development, Saudi Children.

Pak J Med Sci January - March 2008 Vol. 24 No. 1 61-64

INTRODUCTION

Speech and language are tools that humans use to communicate or share thoughts, ideas, and emotions. Language may be expressed through writing, signing, or even gestures in the case of people who have neurological disorders and may depend upon eye blinks or mouth movements to communicate. The most intensive period of speech and language development for humans is the first three years of life, a period when the brain is developing and maturing. Delayed language development (DLD) children may show abnormal findings on functional neuro-radiological investigations, even though structural neuro-radiological studies such as a brain MRI do not show any abnormal findings.¹ DLD constitutes one of the

1. Taha Sadig Ahmed, MD, Ph.D,
2. Abdul Majeed Al Drees, Ph.D,
3. Sultan Ayoub Meo, MBBS, M.Phil, Ph.D,
- 1-3: Department of Physiology, College of Medicine,
King Khalid University Hospital, King Saud University,
4. Gazza Ahmed I, FAAP,
5. Muhammad Al-Nasser M, MRCPCH
- 4-5: Department of Medicine,
King Abdulaziz University Hospital,
Riyadh, Saudi Arabia.

Correspondence

Dr. Taha Sadig Ahmed, Consultant Clinical
Neurophysiologist & Associate Professor,
Dept. of Physiology (29), College of Medicine,
King Khalid University Hospital, King Saud University,
P.O. Box 2925. Riyadh 11461.K.S.A.
E-mail: splendor20@hotmail.com

- * Received for Publication: October 27, 2007
- * Revision Received: December 14, 2007
- * Revision Accepted: December 17, 2007

prominent reasons of children referral to King Abdulaziz University Hospital (KAUH) in Riyadh. The electrophysiological workup of these patients frequently reveals the presence of epileptiform (principally spike/wave) discharges in the EEG, and they occasionally present with a history of epilepsy (defined as two or more unprovoked clinical seizures). Conversely, children attending the hospital with epilepsy frequently have DLD. This raises the possibility of existence of a relationship between epileptiform EEG discharges and delayed language development in children attending KAUH.

An association between EEG abnormalities and language disorders such as Landau-Kleffner syndrome (LKS), continuous spike-wave during slow wave sleep (CSWSS) and atypical benign partial epilepsy is well documented.²⁻⁵ In addition, language deficits can occur in some patients with chronic intractable temporal lobe epilepsy who have mesial temporal sclerosis.⁶ This is explained in terms of neuronal loss and deafferentiation in some of the language-related cortical areas. Moreover, it has recently been shown^{7,8} that prolonged epileptic seizures (status epilepticus) can produce structural changes in the brain and result in deterioration of some mental functions. However, apart from these conditions, it is widely believed that epileptiform discharges, when unaccompanied by clinical seizures, do not warrant medical treatment.⁹

Since the sunup of electroencephalography, it has been held that not every electrical EEG epileptiform discharge should necessarily be accompanied by a clinical seizure. Many epileptiform discharges can occur without external visible clinical manifestations. Such EEG transients were described as "subclinical" or "interictal" or "larval" discharges.¹⁰ Moreover, some spiky EEG events can occur in individuals without epilepsy, although many have other cerebral disease.¹¹⁻¹³ However, it is difficult to envisage recurring interictal epileptiform discharges as being not associated with any effects on normal cerebral functions, on the grounds that they do not cause external clinical manifestations. Considering all these

facts the aim of the present study was to investigate the association between EEG epileptiform discharges and delayed language development in Saudi Children.

PATIENTS AND METHODS

This study was conducted in Department of Physiology and Medicine, College of Medicine, King Khalid University Hospital, King Saud University, Riyadh, Saudi Arabia. The present study was performed on 116 children with DLD who attended the King Abdul Aziz University Hospital (KAUH) during the period 2001-2005. The language problems encountered in these children included expressive language disorders as well as combined expressive-receptive language disorders. Another group of 116 age- & sex-matched normal children served as controls. The controls were consecutive children with a variety of general minor ailments such as common cold, tonsillitis, abdominal pain, without neurological abnormalities, who were referred to Clinical Physiology Laboratory, KAUH for EEG as a part of general workup. The age range in both groups was 2-8 years. Consent was obtained from the parents of the children to be included in the study as long as the names of the children were not to be written or mentioned in public.

Beside general investigations, the children's development was assessed according to the Denver development scale, language evaluated by speech therapist and IQ measured by a psychologist. CT and MRI examination were carried out to exclude any patient with demonstrable cerebral lesion. Moreover, children with history of hypoxic-ischemic injury at birth, meningitis, motor deficit, hearing impairment and IQ <70 were also excluded from the study. Sleep and awake EEG was recorded in each case by using (Compumedics Melbourne, Australia). It included EEG strips undertaken during intermittent photic stimulation and, when the child was old enough to understand instructions, during voluntary hyperventilation. The EEGs were reviewed by a clinical neurophysiologist and a pediatric neurologist. All the EEGs recorded in epileptic patients were

the first EEGs before patients received anti-epileptic medications.

Statistical Analysis: Data were analyzed with SPSS version 15, the frequency of abnormal EEG in both group was compared for statistical significance.

RESULTS

Table-I shows the anthropometric characteristics (Age and Sex) of the pediatric patients. In this study, 84 males and 32 females patients were with DLD and the control group consists of 85 males and 31 females (Table-I). There was no statistically significant difference in terms of age and sex between the groups.

Epilepsy: In the DLD group 14 patients (12.07%) had a history of epilepsy and their EEG showed epileptiform discharges (Table-I). Eleven out of these 14 patients were males (12.94%) of the 85 DLD male patients had seizure. Three out of the 14 patients with seizure were females (9.68%) out of the 31 female DLD patients had seizure. There was no statistically significant difference between the males and females within the DLD group with regard to presence of seizure ($p=0.454$).

While comparing the control group with the DLD patients, there was a significant difference between DLD patients and controls with regard to the presence of seizure. The seizure types within the DLD Group are shown in Table-II. In the control group no subject had a history of seizure.

Abnormal EEG: In the DLD group 26 patients (22.41% out of 116, Table-I) had EEG abnormality (including the above-mentioned 14 patients with epilepsy). Eighteen out of these 26 patients were males, i.e. 21.18% of the 85 DLD male patients had abnormal EEG. Eight out of

Table-II: Seizure types within the DLD group

| Seizure type | No. of patients (%) |
|--|---------------------|
| Generalized Tonic-Clonic Convulsions [GTC] | 5 (35.71) |
| Myoclonic Seizure | 1 (7.14) |
| Absence Seizure | 1 (7.14) |
| Temporal lobe Seizure | 1 (7.14) |
| Frontal lobe Seizure | 2 (14.3) |
| Occipital lobe Seizure | 1 (7.14) |
| Benign Childhood Epilepsy with Centrotemporal Spikes [BECTS] | 3 (21.4) |

the 26 patients with abnormal EEG were females, i.e. 25.81% out of the 31 female DLD patients had abnormal EEG. There was no statistically significant difference in the prevalence of EEG abnormalities between the males and females within the DLD group ($p=0.383$). However, in the control group one male (0.86% out of 116) had EEG abnormality in the form of a right central spike which appeared only during sleep (Table-I).

When comparing the control group with the DLD patients, it became clear that DLD patients had a significantly higher percentage of abnormal EEGs (22.41%) compared to normal children (0.86%), $p<0.001$. Out of the 102 DLD patients who had no seizure, 12 patients (11.76%) of them had abnormal EEG. These results do not include a case of Landau-Kleffner syndrome or continuous spike-wave during slow wave sleep (CSWSS).

DISCUSSION

Delayed language development in children is an important public health problem. Speech problems may include stuttering or dysfluency, articulation disorders, or unusual voice quality. Several types of speech and language delay and disorders have been described, Express-

Table-I: Comparison between the controls and DLD patients in terms of sex, age, EEG Epileptiform discharges and Epilepsy.

| | Controls (n=116) | | DLD patients (n=116) | | Statistical significance |
|--|------------------|-------------|----------------------|-----------|--------------------------|
| | Male | Female | Male | Female | |
| Sex | 84 | 32 | 85 | 31 | NS |
| Age (Mean±SD) | 4.07±1.12 | 4.01±1.13 | 4.03±1.11 | 3.95±1.05 | NS |
| Epileptiform EEG discharges | 1 (0.86%) | 26 (22.41%) | $P<0.001$ | | |
| Epilepsy | 0 | 14 (12.07%) | $P<0.001$ | | |
| No history of seizure but epileptiform discharges present in EEG | 1 (0.86%) | 12 (11.76%) | $P<0.001$ | | |

sive language delay may exist without receptive language delay, but they often co-occur in children. Some children also have disordered language. These language problems can involve difficulty with grammar (i.e., syntax); words or vocabulary (semantics); the rules and system for speech sound production (phonology); units of word meaning (morphology); and the use of language, particularly in social contexts (pragmatics).¹⁴ The present study was carried out to investigate the possible relationship between EEG abnormalities and delayed language development in Saudi children who had no mental retardation, hearing impairment or radiological evidence of cerebral lesion.

The results obtained in the current study show that children with DLD had significantly higher percentage of abnormal EEGs (22.41%) than control children (0.86%). Similarly, DLD children had a high percentage of epileptic syndromes (12.07%), whereas none of the control children had epilepsy.

Those children with language disorders have increased frequency of both EEG abnormalities and clinical seizure (epilepsy) has been noted by other investigators as well.^{15,16} With regard to epilepsy Dalby¹⁴ reported it in up to 20% of children with language disorders, and Tuchman et al¹⁵ reported it in 8% of these children. Overall, our results, in support of Dalby¹⁴ and Tuchman et al,¹⁶ confirm an association between epilepsy and language disorders.

Moreover, Tuchman et al¹⁶ reported epileptiform discharges in 9% of children with language disorder who had no epilepsy. We found similar rate of epileptiform discharges in 11.76% of such children. Both studies show a clear association between epileptiform discharges and language disorders. It is our contention that repetitive subclinical epileptiform discharges on their own, even in patients without epilepsy, can be interfere with normal language development.

CONCLUSIONS

Keeping in view the results of the present study it is concluded that the children with delayed language development are associated

with higher prevalence of epileptiform EEG discharges and epilepsy compared to their control group.

REFERENCES

1. Im SH, Park ES, Kim DY, Song DH, Lee JD. The neuroradiological findings of children with developmental language disorder. *Yonsei Med J* 2007;48(3):405-11.
2. Aicardi J. Epileptic encephalopathies of early childhood. *Curr Pin Neurol Neurosurg* 1992;5:344-8.
3. Lanzi G, Veggiotti P, Contes S, Partesana E, Resi C. A correlated fluctuation of language and EEG abnormalities in a case of the Landau-Kleffner syndrome. *Brain Dev* 1994;16:329-34.
4. Parry-Fielder B, Nolan TM, Collins KJ, Stojcevski Z. Developmental language disorders and epilepsy. *J Paediatr Child Health* 1997;33:277-80.
5. Praline J, Hommet C, Barthez MA, Brault F, Perrier D, Passage GD, et al. Outcome at adulthood of the continuous spike-wave during slow sleep and Landau-Kleffner syndrome. *Epilepsia* 2003;44:1434-40.
6. Bartha L, Benke T, Bauer G, Trinka E. Interictal language functions in temporal lobe epilepsy. *J Neurol Neurosurg Psychiatry* 2005;76:808-14.
7. Szabo K, Poepl A, Pohlmann-Eden B, Hirsch J, Back T, Sedlaczek O, et al. Diffusion-weighted and perfusion MRI demonstrates parenchymal changes in complex partial status epilepticus. *Brain* 2005;128:1369-76.
8. Young GB. Status epilepticus and brain damage: Pathology and pathophysiology. *Adv Neurol* 2006;97:217-20.
9. Binnie CD. Cognitive impairment during epileptiform discharges: is it ever justifiable to treat the EEG? *The Lancet Neurology* 2003;2:725-30.
10. Gibbs FA, Lennox WG, Gibbs EL. The electroencephalogram in diagnosis and in localization of epileptic seizures. *Arch Neurol Psychiatry* 1936;36:1225-35.
11. Zivin L, Ajmone MC. Incidence and prognostic significance of "epileptiform" activity in the EEG of non-epileptic subjects. *Brain* 1968;91:751-8.
12. Hughes J. EEG in clinical practice. Boston: Butterworth, 1994;165-81.
13. Echenne B, Cheminal R, Rivier F, Negre C, Touchon J, Billiard M. Epileptic electroencephalographic abnormalities and developmental dysphasias: A study of 32 patients. *Brain Dev* 1992;14:216-25.
14. Mabry IR. Screening for speech and language delay in preschool children. *Am Fam Physician* 2006;74(8):1373-4.
15. Dalby M. Aetiological studies in language retarded children. *Neuropaediatrics* 1977;8(suppl):499.
16. Tuchman R, Rapin I, Shinnar S. Autistic and dysphasic children. II. Epilepsy. *Pediatrics* 1991;88:1219-25.