**Electroencephalographic Findings In Children With Autism Spectrum Disorder**

**By**

**Shimaa Abdallah Elsayed Ahmed**

MB.B.CH

**Under Supervision Of**

|  |  |
| --- | --- |
| **Prof. Khaled Hussein Taman** | **Prof. Samia Samy Aziz**  |
| Professor Of PediatricsMedical Studies DepartmentInstitute Of Postgraduate Childhood StudiesAin Shams University | Professor Of Child Mental HealthMedical Studies DepartmentInstitute Of Postgraduate Childhood StudiesAin Shams University |
| **Dr. Khaled Ossama Abdbulghani**Lecturer Of Neurology And PsychiatryFaculty Of MedicineHelwan University |

**Abstract**

Autism Spectrum Disorder is a neurodevelopmental disorder characterized by impairments in social communication, reciprocal social interaction, and repetitive behaviors and interests. It was previously known as Pervasive Developmental Disorders. It affect 1 in 88 children, Males are affected four times more than females. It has a complex and multifactorial aetiology. It is known to be highly heritable. It is frequently associated with comorbid psychopathology as high as 70%. The most common are intellectual disability, ADHD, Eating disorder, depression, sleep disorder and Anxiety disorder. There is no ‘‘gold standard’’ measure for assessing ASD so Diagnosis takes place typically from a complete history, physical and neurological evaluation. EEG has been the primary measure used to capture and characterize epileptiform and abnormal paroxysmal activity through the detection of focal spikes, which occur with increased frequency in ASD.

**Methods:** Crosssectional descriptive study, conducted on 32 children attending the outpatient clinic of Special Need Center, Institute of Postgraduate Childhood studies, Ain Shams University.They underwent Thorough Full medical history, clinical examination, Clinical Psychiatric assessment using CARS, IQ test and EEG.

**Results:** ASD is more common in males than females, although 53.1% had positive history of consanguinity but no statistically significant difference. As regarding EEG findings, 56.3% of children had normal EEG Finding; while 43.8% had abnormal EEG Findings. 50% with abnormal EEG Findings had subcortical Dysrythmia, 14.3% Generalized Epileptic Dysrythmia. There was no statistically significant relationship between different EEG Findings and CARS in the studied children with ASD.

**Conclusion:** ASD is a neurodevelopmental disorder with altered brain connectivity. There is no agreement on EEG features in ASD. Although clinical EEG studies generally agree on the high prevalence of epileptiform abnormalities in children with ASD.

**Key words:** Autism Spectrum Disorder, Electroencephalograhy, EEG.

**Introduction**

Autism spectrum disorder (ASD) is a lifelong neurodevelopmental disorder that is often diagnosed during early childhood and is characterized by impairments in social communication, reciprocal social interaction, and repetitive or restricted behaviors and interests **(APA, 2013).** It had a complex aetiology However; an etiological factor has been identified in only 15–20% of persons with ASD **(Schaefer and Mendelsohn, 2008).** Typical diagnosis occurs at 3–4 years of age **(Matson et al., 2008).**

It is frequently associated with intellectual impairment, structural language disorder, psychiatric symptoms, ADHD, developmental coordination disorder ,anxiety disorders, depressive disorders, specific learning difficulties, medical conditions, avoidant-restrictive food intake disorder **(APA, 2013).**

 Electroencephalography is defined as electrical activity recorded from the scalp surface by metal electrodes and conductive media **(Niedermeyer and Lopes da Silva, 1993).** it is used to assess functional connectivity between different brain regions over time via EEG coherence, and quantitative measurement of the relationship of frequency spectra between two EEG signals **(Olejiczak, 2009).**

Many studies suggested that ASD was a connectivity disorder **(Assaf et al., 2010).** While a number of previous studies have reported underconnectivity in ASD cortex **(Coben et al., 2008),** others had indicated mixed or overconnectivity **(Shih et al., 2011)** and/or aberrant lateralization in ASD brain connectivity **(Lee et al., 2009).**

**AIM OF THE STUDY**

1. To assess EEG findings in children with autism spectrum disorder.
2. To correlate between different levels of Autism Spectrum Disorder and EEG findings.

**Subjects And Methods**

**Type Of The Study:** CrossSectional Descriptive Study

**Subjects:** The study was conducted on 50 children diagnosed as ASD according to DSM V. The children attended the outpatient clinic of Special Need Center, Institute of Postgraduate Childhood, Ain Shams University from the period January 2014 till July 2015. The study enrolled only 32 children because of lack of detailed data or dropping out during the study.

**Inclusion Criteria**

1. Age: from age of (3-10 years).
2. Gender: both sexes.
3. Cases diagnosed as Autism Spectrum Disorder according to DSM-v.
4. **Exclusion Criteria**
5. Children with autism spectrum disorder and epilepsy.
6. Children suffering from chronic medical diseases.
7. Children with other neurological or psychiatric diseases.

**Methods**

**All children were subjected to the following:**

1. **Full medical history** Focusing on age of onset, Course and duration of disease – Symptoms of ASD - Severity of symptoms - Prenatal, natal and postnatal history - Developmental history –Family and past history - History of consanguinity.
2. **Thorough clinical examination** focusing on neurological assessment.
3. **Clinical Psychiatric interview**: All children were diagnosed according to DSM-V criteria of ASD **(APA, 2013).**
4. **Assessment of children through**:
5. **Childhood Autism Rating Scale** (**CARS)(Schopler et al., 1986)**

It is a behavior rating scale help to diagnose ASD and Assessment of severity of autistic symptoms **(Ozonoff et al., 2011).** It rates the child on a scale from 1 to 4 in each of 15 areas. It is completed by a clinician based upon observations and/or caregiver reports. Intellectual ability and language level are included as part of the total score **(Hong et al., 2011).**

 The possible range of scores is 15–60. A total score classifies children’s behavior as having no autism (15–29,5), mild to moderate autism (30–36.5) and severe autism (37–60) **(Rellini et al., 2004).**

**Electroencephalography (EEG):**

It is done using (compumedics- E series Device- Type 5CP70 – Serial J11D725). It is a fully digital amplifier system with a standard network interface that is compatible with most current personal computers. With 22 channel configurations, these amplifier systems are appropriate for both full sleep and EEG data collection.

**c. IQ test using Stanford-Binet Intelligence Scales-fivthEdition, The Arabic version (أبو النيل ,2011)**

It is used to assess intellectual ability between ages 2 and 16 years. It consists of 10 subscales. The three areas assessed are general cognitive functioning, verbal and nonverbal intelligence and five factors formed into groups along verbal\ nonverbal measures: fluid reasoning, knowledge, quantative reasoning, visual-spatial processing, and working memory together **(Roid, 2003).**

**LIMITATION OF THE STUDY**

The study conducted on about 50 children many of them excluded due to

1. Refusal of parents of children to participate in the study.
2. Incomplete data as regards EEG which could be defective or in apparent.

**ETHICAL ASPECT**

Informed written consent was obtained from parents after explanation of the aim of the study and its benefits.

**Results**

**Table (1): Demographic characteristics of the studied Children with ASD (n=32)**

| **Sex** | **n** | **%** | **x2** | **P-value** |
| --- | --- | --- | --- | --- |
| Male | 18 | 56.3 | 0.500 | 0.480 |
| Female | 14 | 43.8 |
| **Age** | **Mean + SD** | **Min.** | **Max.** | **Range** |
| 5.30 + 1.96 | 3.0 | 10.0 | (3.0-10.0) |

 As shown in table (1). The sample ages range from (3-10) years and it affects males more than female as shown in table

**Figure (1): Previous Medical History of studied children with ASD**

As shown in figure (1) (53.1%) of the studied children had positive history of consanguinity; (40.7%) due to Neonatal and infantile causes, (25.0%) had no previous medical history, (15.6%) had maternal causes.

**Table (2): EEG Findings in the studied patients with ASD (n=32)**

| **EEG Finding** | **n** |  **%** |
| --- | --- | --- |
| Normal EEG Finding | 18 | 56.3 |
| Abnormal EEG Finding | 14 | 43.8 |

**Figure (2): Types of Abnormal EEG Findings**

As shown in table (2) (56.3%) of children had normal EEG; while (43.8%) had abnormal EEG Findings. The abnormal EEG findings were (50%) had sub cortical Dysrythmia, (14.3%) Generalized Epileptic Dysrythmia, (7.1%) Immature ECG finding, Anterior Temporal Dysrythmia, Bilateral Centro temporal Dysrythmia, Left Frontal Dysrythmia and Left front central Dysrythmia with tendency for generalization as shown in figure (2).

**Table (3): Description of IQ and CARS in Children with ASD (n=32)**

| **IQ** | **Mean + Sd** | **Min.** | **Max.** | **Range** |
| --- | --- | --- | --- | --- |
| 65 + 10 | 40 | 78 | (40-78) |
| **CARS** | **Mean + Sd** | **Min.** | **Max.** | **Range** |
| 32.2 + 3.2 | 29.50 | 45.0 | (29.50-45.0) |

 As shown in table (3) the mean IQ of children was (65) and The mean CARS was (32.2 + 3.2).

**Figure (3): Symptomatology in the studied Children with ASD (n=32)**

 As shown in figure (3) (40.6%) of children had Language Regression, (40.6%) were hyperactive, (15.6%) had Echolalia, Poor social interaction, (18.8%) had sensory dysfunction, disturbed eating, (9.4%) had symptomatology of ASD only, (6.3%) had disturbed sleep, (3.1%) experienced self-Injurious behavior, poor attention, aggression.

**Table (4): CARS in children with different EEG findings (n=32)**

| **EEG Finding**  | **CARS** | **x2** | **P-value** |
| --- | --- | --- | --- |
| Normal | **Mean + Sd** |
| 31.9 + 3.6 | 1.192 | 0.344 |
| Immature | **Mean + Sd** |
| 29.5 + 0.0 |
| Generalized Epileptic Dysrythmia | **Mean + Sd** |
| 35.0 + 0.0 |
| Anterior Temporal Dysrythmia | **Mean + Sd** |
| 33.3 + 0.9 |
| Bilateral Centrotemporal Dysrythmia | **Mean + Sd** |
| 34.0 + 0.0 |
| Left Frontal Dysrythmia | **Mean + Sd** |
| 38.9+ 0.0 |
| Subcortical Dysrythmia | **Mean + Sd** |
| 31.0 + 1.3 |
| Left frontocentral Dysrythmia with tendency for generalization | **Mean + Sd** |
| 36.0 + 0.0 |

As shown in table (4) Left Frontal Dysrythmia had got the highest CARS (38.9), Left frontocentral Dysrythmia with tendency for generalization was (38.9), while Immature, Normal and subcortical dysthrymia (29.5, 31.9, 31.0) respectively

**Discussion**

The number of reported cases of ASD increased dramatically in last years. This increase is attributable to changes in diagnostic practices, referral patterns, availability of services, age at diagnosis, and public awareness **(Chawarska andVolkmar, 2005)**. The clinical signs of ASD are known to emerge concurrently with a period of abnormal brain and head “overgrowth” occurring within approximately the first year of life and plateaus into adulthood **(Hazlett et al., 2011)**.Our results pointed to the higher risk of ASD in boys than girls about (56.3%) of the studied children were males, while (43.8%) were females and there was no statistically significant difference between the studied children with ASD as regard their ages (P>0.05).This finding was consistent with that reported by **Itzchak et al (2010)** who found 461 children (81%) out of 564 participants were male autistic patients. **Fombonne (2009)** showed a marked male preponderance, with the male-to-female ratio ranging 4:1. Another study conducted from a pediatric hospital at Ain Shams University and found that boys had higher risk of autism than girls **(El-Baz et al., 2011)**. **Levy et al (2009)** found that Males are affected four times more frequently than females.

The causes of ASD are still unclear, although results from twin and family studies provide evidence for a strong genetic contribution **(Paul et al., 2010)**. Despite significant research on prenatal, perinatal, neonatal, and other risk factors in autism, the causal nature of these associations is still disputed due to several current methodological limitations of studies **(Gardener et al., 2009)**. It is known to be highly heritable with a recurrence rate of 19% in siblings **(Ozonoff et al., 2011)**.

We found that (53.1%) had positive history of consanguinity but there was no statistical significance but **El baz et al (2011)** found that Positive family history was found to be significantly associated with the risk of ASD (16% of cases versus 1% of controls).Similar findings was reported by **Bilder et al (2009)**.

Higher prevalence of ASD also had been associated with obstetric and neonatal factors that result in NICU admission **(Kuban et al., 2009**). **Schendel and Bhasin(2008)** found a twofold increase in ASD risk as a result of lower birth weight and gestational age. **El baz et al (2011)** found a history of low birth weight and using instrumental tools during delivery were significantly higher in cases than controls. Postnatal factors as history of hypoxia, resuscitation, history of neonatal jaundice were also statistically significantly increased in autistic patients**. Kolevzon et al (2007)** suggested the presence of non-heritable prenatal and perinatal risk factors for autism. **Bolton et al (1992)** demonstrated an association between autism and obstetric complications, prenatal or intrapartum use of medications.

The mean IQ of the studied children was (65) as most of them had mild to severe retardation, **as El Baz et al (2011)** found that 55% of children had mild to severe retardation and **Baron- Cohen et al (2006)** who reported that autistic children had spectrum of IQ ranged from 0 to 60.

There is no ‘‘gold standard’’ measure for assessing ASDs **(Kleinman et al., 2008)**; however, best practice involves utilizing various methods of garnering information including interviews, observation, and rating scales that involve multiple informants such as parents, teachers, and alternative caregivers **(Haynes and O’Brien, 2000)**.

Core diagnostic features are evident in the developmental period but Manifestations of the disorder also vary depending on the severity of the autistic condition, developmental level, and chronological age; hence, the term spectrum**(APA, 2013)***.* In this study, as regarding symptomatology of ASD about (40.6%) of the studied children had Language Regression, (15.6%) had Echolalia, (18.8%) had sensory dysfunction, (9.4%) had symptomatology of ASD only. **El- baz et al (2011)** found that (72) of children presented with delayed speech, (11%) play alone, (9%) inattention, and (8%) with loss of eye contact.

Also, we found that (3.1%) experienced self injurious behavior, (3.1%) had inattention, (3.1%) were aggressive.

Maladaptive behaviors frequently associated with ASD include hyperactivity/inattention, aggression, and motor stereotypies. Prior studies have also shown that ID is a strong predictor of greater severity in ASD **(Matson and Shoemaker, 2009)** and is associated with various mala­daptive behaviors **(Emerson et al., 2001).**

Also, (40.6%) of children were hyperactive. **Sturm et al (2004)** found that ADHD symptoms are present in about 20–80% of children with ASD. Also, the severity of ASD correlates with the co-occurrence of ADHD symptoms **(Holtmann et al., 2007)**. Impairment of motor control, including neurological soft signs is common in ASD **(Price et al., 2012)**.

In this study, (9.4%) had Delayed Milestones. **El baz et al (2011)** found that all studied developmental milestones were delayed in autistic children than control group. The difference was statistically significant. **Mc Partland (2006)** found that children with autism may be delayed in acquiring motor activity.

Also, we found that (18.8%) had disturbed eating. Children with ASD were found to have significantly more feeding problems and eat significantly a narrower range of foods than children without ASD which may be due to food allergies or intolerances or from autistic features **(Schreck et al., 2004)**.

We found that (6.3%) had sleep disorders. **Krakowiak et al (2008)** found that Sleep problems are present in 80% of children with ASD. **Rzepecka et al (2011)** found that 77.2% of children with ASD had sleep problems. **Mannion et al (2013)** found that 80.9% of children and adolescents with ASD presented with sleep problems. **Mayes and Calhoun (2009)** found that sleep problems were not related to age, IQ, gender, race, parent occupation, neuropsychological functioning and learning ability and increased with severity of autistic symptoms and with severity of parent reported symptoms.

we found that (40.6%) of the studied children had Language Regression which is defined as a period of normal development followed by a significant change in which there is a loss of previously acquired language and other skills, this is in accordance with **Baird et al (2008)** that found Regression occurs in 15–40% of children with autism but **Luyster et al (2005)** found that this pattern associated with about a quarter of the ASD population.

Also, we found (43,8%) of children had abnormal EEG most commonly subcortical Dysrythmia and Generalized Epileptic Dysrythmia. **El baz et al (2011)** had 31% of autistic children epileptic focus in EEG, with and without a history of convulsions. **Ballaban and Tuchman (2000)** found that 64 patients with autism out of 316 children evaluated for ASD had EEG findings. These findings confirm the importance of ongoing medical follow-up for children with ASDS, especially for those with abnormal EEG results. Also, **Kanemura et al (2013)** found that EEG paroxysmal abnormalities were present in 11 to 21 patients (52.4%).

**Chez et al (2006)**, found that the incidence of abnormal epileptiform activity on EEG is high as many as 60–75% of individuals with autism. Epileptiform activity itself might contribute to dysfunction of language and social regions of the brain **(Ballaban and Tuchman , 2000)**.

As regarding relationship between different EEG Findings and CARS in there was no statistically significance although children with left frontal dysrythmia had got the highest CARS (38.9), and left frontocentral dysrythmia with tendency for generalization was (36).

**Conclusion:** ASD is a neurodevelopmental disorder with no agreement on the EEG features of ASD. Although clinical EEG studies generally agree on the high prevalence of epileptiform abnormalities in children with ASD**.**

**Recommendation**

1. Early detection of abnormalities in EEG signals may allow early intervention to prevent or ameliorate lifelong conditions.
2. Long term EEG is required is required to allow better findings and events.
3. Follow up of children with normal EEG is required to detect any changes.
4. **Reference**
5. **Alexander, A. L., Lee, J. E., Lazar, M., Boudos, R., DuBray, M. B., Oakes, T. R., et al (2007):** Diffusion tensor imaging of the corpus callosum in Autism, Neuroimage 2007, 34: 61-73.
6. **American Psychiatric Association (2013):** Diagnostic and statistical manual of mental disorders (5th ed.). Washington, DC: Author.
7. **Assaf, M., Jagannathan, K., Calhoun, V. D., et al (2010):** Abnormal functional connectivity of default mode sub-networks in autism spectrum disorder patients, Neuroimage; 53: 247–256.
8. **Ballaban-Gil K, Tuchman R. Epilepsy and epileptiform EEG (2000):** association with autism and language disorders, Ment Retard Dev Disabil Res Rev; 6: 300–308.
9. **Baron-Cohen S, Hoekstra RA, Knickmeyer R, et al (2006):** The autism spectrum quotient (AQ): adolescent version. J Autism Dev Discord; 36: 343–350.
10. **Bilder D et al. (2009):** Prenatal, perinatal and neonatal factors associated with autism spectrum disorders. Pediatrics; 123: 1293.
11. **Bolton P, Pickles A, Harrington R, et al. (1992):** Season of birth: issues, approaches and findings for autism. J Child Psychol Psychiatry; 33: 509–530.
12. **Casanova, M. F., van Kooten, I. A. J., Switala, A. E., van Engeland, H., Heinsen, H., Steinbusch, H. W. M., et al. (2006):** Minicolumnar abnormalities in autism. Acta Neuropathologica; 112: 287–303.
13. **Casanova, M., Trippe, J. (2009):** Radial cytoarchitecture and patterns of cortical connectivity in autism. Philos Trans R Soc Lond B Biol Sci.; 364: 1433-1436.
14. **Center for Disease Control and Prevention (2012):** Prevalence of autism spectrum disorders; 61: 1–19.
15. **Chez MG, Chang M, Krasne V, Coughlan C, Kominsky M, Schwartz A.(2006):** Frequency of epileptiform EEG abnormalities in a sequential screening of autistic patients with no known clinical epilepsy from 1996 to 2005. Epilepsy Behav.; 8: 267–271.
16. **Coben, R., Clarke, A. R., Hudspeth, W., Barry, R. J.(2008):** EEG power and coherence in autistic spectrum disorder. Clinical Neurophysiology; 119: 1002–1009.
17. **El-Baz F, Ismael NA, Nour Eldin SM. (2011):** Risk factors for autism: An Egyptian study. Egyptian Journal of Medical Human Genetics; 12: 31–38.
18. **Emerson E, Kiernan C, Alborz A, et al.** **(2001):** The prevalence of challenging behaviors: a total population study*. Research in* Developmental Disabilities; 22: 77–93.
19. **Fombonne, E. (2009):** Epidemiology of pervasive developmental disorders. Pediatric Research ; 65: 591–598.
20. **Gardener H, Spiegelman D, Buka SL. (2009):** Prenatal risk factors for autism: comprehensive meta-analysis. Br J Psychiatry;195: 7–14.
21. **Haynes, S. N., O’Brien, W. H.(2000):** Principles and practice of behavioral assessment. New York: Plenum Publishing Corporation .
22. **Hazlett HC, Poe M, Gerig G, Smith RG, Provenzale J, Ross A, et al. (2005):** Magnetic resonance imaging and head circumference study of brain size in autism: birth through age 2 years. Arch Gen Psychiatry; 62: 1366–1376.
23. **Hazlett, H.C., Poe, M.D., Gerig, G., Styner, M., Chappell, C., Smith, R.G., Vachet, C., Piven, J. (2011):** Early brain overgrowth in autism associated with an increase in cortical surface area before age 2 years. Arch. Gen. Psychiatry; 68: 467–476.
24. **Holtmann, M., Bolte, S., Poustka, F.(2007):** Attention deficit hyperactivity disorder symptoms in pervasive developmental disorders: Association with autistic behavior domains and coexisting psychopathology. Psychopathology; 40: 172–177.
25. **Hong S, Ke X, Tang T, Hang Y, Chu K, Huang H, et al. (2011):** Detecting Abnormalities of corpus callosum connectivity in autism using magnetic resonance imaging and diffusion tensor tractography. Psychiatry Research; 194: 333–339.
26. **Hughes JR. (2007):** Autism: the first firm finding underconnectivity? Epilepsy Behav ; 11: 20–24.
27. **Ijichi S, Ijichi N. (2004):** The prenatal autistic imprinting hypothesis: developmental maladaptation to the environmental changes between womb and the social world. Med Hypotheses; 62: 188-194.
28. **Itzchak EB, Zachor DA, Assaf Harofeh. (2010):** Male:female ratio is related to autism spectrum disorder in the family and to maternal age. Harofeh Medical Center, Zerifin, Israe. Franklin Hall B Level 4 (Philadelphia Marriott Downtown l).
29. **Kana, R.K., Keller, T.A., Cherkassky, V.L., Minshew, N.J., Just, M.A. (2006):** Sentence comprehension in autism: thinking in pictures with decreased functional connectivity. Brain; 129: 2484–2493.
30. **Kanemura H, Sano F, Tando T, Sugita K, Aihara M. (2013):** [Can  EEG  characteristics predict development of epilepsy in autistic children](http://www.sciencedirect.com.ugrade1.eul.edu.eg:2048/science/article/pii/S1090379812002218)?, European Journal of Paediatric Neurology;17: 232-237*.*
31. **Kleinhans, N.M., Richards, T., Sterling, L., Stegbauer, K.C., Mahurin, R., Johnson, L.C., Greenson, J., Dawson, G., Aylward, E. (2008):** Abnormal functional connectivity in autism spectrum disorders during face processing. Brain; 131: 1000–1012.
32. **Kleinman, J. M., Ventola, P. E., Pandey, J., Verbalis, A. D., Barton, M., Hodgson, S., et al. (2008):** Diagnostic stability in very young children with autism spectrum disorders. Journal of Autism and Developmental Disorders ; 38: 606–615.
33. **Kolevzon A, Gross R, Reichenberg A. (2007):** Prenatal and perinatal risk factors for autism: a review and integration of findings. Arch Pediatr Adolesc Med.; 161: 326–333.
34. **Krakowiak P, Goodlin-Jones B, Hertz-Picciotto I, Croen LA, Hansen RL. (2008):** Sleep problems in children with autism spectrum disorders, developmental delays, and typical development: a population-based study. J Sleep Res.; 17: 197–206.
35. **Kuban KC, O’Shea TM, Allred EN, Tager-Flusberg H, Goldstein DJ, Leviton A. (2009):** Positive screening on the Modified Checklist for Autism in Toddlers (M-CHAT) in extremely low gestational age newborns. J Pediatr.; 154: 535–540.
36. **Lee, P.S., Yerys, B.E., Della Rosa, A., Foss-Feig, J., Barnes, K.A., James, J.D., VanMeter, J., Vaidya, C.J., Gaillard, W.D., Kenworthy, L.E. (2009):** Functional connectivity of the inferior frontal cortex changes with age in children with autism spectrum disorders: fcMRI study of response inhibition. Cerebral Cortex; 19: 1787–1794.
37. **Levy, S. E., Mandell, D. S., Schultz, R. T. (2009):** Autism. Lancet; 374: 1627–1638.
38. **Luyster, R., Richler, J., Risi, S., Hsu, W.-L., Dawson, G., Bernier, R., et al. (2005):** Early regression in social communication in autistic spectrum disorders: a CPEA study. Developmental Neuropsychology; 27: 311–336.
39. **Mannion, A., Leader, G., Healy, O. (2013):** An investigation of comorbid psychological disorders, sleep problems, gastrointestinal symptoms and epilepsy in children and adolescents with autism spectrum disorder. Research in Autism Spectrum Disorders; 7: 35–42.
40. **Mason, R. A., Williams, D. L., Kana, R. K., Minshew, N., Just, M. A.(2008):** Theory of Mind disruption and recruitment of the right hemisphere during narrative comprehension in autism. Neuropsychologia; 46: 269-280.
41. **Matson JL and Shoemaker M. (2009):** Intellectual disability and its relationship to autism spectrum disorders. Research in Developmental Disabilities; 30: 1107–1114.
42. **Matson, J. L., Wilkins, J., Gonza´ lez. (2008):** Early identification and diagnosis of autism spectrum disorders in young children and infants: How early is too early? Research in Autism Spectrum Disorders; 2: 75–84.
43. **Mayes, S. D., Calhoun, S. L. (2009):** Variable related to sleep problems in children with autism. Research in Autism Spectrum Disorders; 3: 931–941.
44. **McAlonan, G.M., Cheung, V., Cheung, C., Suckling, J., Lam, G.Y., Tai, K.S., Yip, L., Murphy, D.G., Chua, S.E. (2005):** Mapping the brain in autism. Avoxel-based MRI study of volumetric differences and intercorrelations in autism. Brain; 128: 268–276.
45. **McPartland PJ, Klin A. (2006):** Asperger’s syndrome. Adolesc Med Clin; 17: 771–788.
46. **Niedermeyer, F. H. Lopes da Silva. (1993):** Electroencephalography: Basic principles, clinical applications and related fields, 3rd edition, Lippincott, Williams, Wilkins, Philadelphia.
47. **OlejniczakP.(2009):** **Neurophysiologic basis of EEG.**JClinNeurophysiol*;* 23: 186-189.
48. **Ozonoff, S., Young, G.S., Carter, A., Messinger, D., Yirmiya, N., Zwaigenbaum, L., et al. (2011):** Recurrence risk for autism spectrum disorders: a baby sibling's research consortium study. Pediatrics; 128: 488–495.
49. **Paul L, Eva C, Maria C, Christopher G, Henrik A. (2010):** The genetics of autism spectrum disorders and related neuropsychiatric disorders in childhood. Am J Psychiatry; 167: 30.
50. **Price, K. J., Shiffrar, M., Kerns, K. A.(2012):** Movement perception and movement production in Asperger’s syndrome. Research in Autism Spectrum Disorders; 6: 391–398.
51. **Rellini E, Tortolani D, Trillo S, Carbone S, Montecchi F.(2004):** Childhood Autism Rating Scale (CARS) and Autism Behavior Checklist (ABC) correspondence and conflicts with DSM-IV criteria in diagnosis of autism. J Autism Dev Disord; 34: 703–708.
52. **Roid, G.H. (2003):** Stanford–Binet intelligences cales(SB5), Rolling Meadows,IL:Riverside.
53. **Rzepecka, H., McKenzie, K., McClure, I., Murphy, S. (2011):** Sleep, anxiety and challenging behaviour in children with intellectual disability and/or autism spectrum disorder. Research in Developmental Disabilities; 2758–2766 .
54. **Schendel DE, Bhasin TK. (2008):** Birth weight and gestational age characteristics of children with autism, including a comparison to other developmental disabilities. Pediatrics; 121: 1155–1164.
55. **Schopler E, Reichler RJ, Renner BR. (1986):** The childhood autism rating scale (CARS): for diagnostic screening and classification of autism.
56. **Schreck, K. A., Williams, K., Smith, A. F. A. (2004):** comparison of eating behaviors between children with and without autism. Journal of Autism and Developmental Disorders; 34: 433–438.
57. **Shih, P., Keehn, B., Oram, J.K., Leyden, K.M., Keown, C.L., Muller, R.A. (2011):** Functional differentiation of posterior superior temporal sulcus in autism: a functional connectivity magnetic resonance imaging study. Biological Psychiatry; 70: 270–277.
58. **Sturm, H., Fernell, E., Gillberg, C.(2004):** Autism spectrum disorders in children with normal intellectual levels: Associated impairments and subgroups. Developmental Medicine and Child Neurology; 46: 444–447.

محمود السيد أبو النيل (2011), مقياس ستانفورد بينيه للذكاء- الصورة الخامسة- المؤسسة العربية لاعداد وتقنين ونشر الاختبارات النفسية, القاهرة, مصر.

**نتائج رسم المخ الكهربائى المصاحبه لاضطراب طيف التوحد فى الأطفال**

**مقدمة من**

**الطبيبه / شيماء عبد الله السيد أحمد**

بكالوريوس الطب والجراحه

**تحت إشراف**

|  |  |
| --- | --- |
| **أ.د/ خالد حسين طمان** | **أ.د/ساميه سامى عزيز** |
| أستاذ طب الأطفالقسم الدراسات الطبيةمعهد الدراسات العليا للطفولةجامعة عين شمس | أستاذ صحة الطفل العقليةقسم الدراسات الطبيةمعهد الدراسات العليا للطفولةجامعة عين شمس |
| **د. خالد أسامه عبد الغنى**مدرس أمراض المخ و الأعصاب والطب النفسىكلية الطبجامعة حلوان |

**المستخلص**

يعد اضطراب طيف التوحد أحد الأضطرابات النمائيه ويتم تشخيصه خلال مرحلة الطفولة المبكرة ويتميز بالعجز المستمر في التواصل و التفاعل الاجتماعي عبر سياقات متعددة، ويشمل الاتصال اللفظى وغير اللفظى المستخدم، وتتمثل نسبته بين الأطفال 1 لكل 88 طفل، ويتأثر الذكور أربع مرات أكثر من الإناث. و المسببات المرضية له معقدة وليست محدده. ومن المعروف أنه وراثيا وكثيرا ما يرتبط بأمراض نفسيه آخرى بنسبه تصل الى70٪ وتشمل الإعاقة الذهنية، فرط الحركه وتشتت الأنتباه، اضطرابات الأكل، والاكتئاب، واضطراب النوم واضطراب القلق. ليس هناك 'معيار ذهبى أو مقياس لتشخيص التوحد ولكن التشخيص عادة من خلال التاريخ المرضى الكامل والفحص الأكلينيكى والتقييمات النفسيه. ويعتبر رسم المخ الكهربائى المقياس الأساسي المستخدم لالتقاط وتمييز أنشطة المخ غير الطبيعية.

**منهج البحث:** الدراسة وصفية مقطعية، أجريت على 32 طفلا حضروا العيادة الخارجية بمركز ذوى الأحتياجات الخاصه، معهد الدراسات العليا للطفولة، جامعة عين شمس.وقد خضع المرضى للتاريخ المرضى الطبي والفحص السريري والتقييم النفسي باستخدام اختبار كارز و رسم المخ.

**النتائج:** مرض طيف التوحد أكثر شيوعا فى الذكور حيث أن 56.3٪ من الأطفال فى العينه ذكور ،43.8٪ من الإناث، كما أن53.1٪ لديهم قرابة بين الأبوين ولكن لا يوجد فرق ذو دلالة إحصائية. وأما نتائج رسم المخ كان 56.3٪ من الأطفال لديهم رسم مخ طبيعى و 43.8٪ لديهم رسم مخ غير طبيعي. وكان متوسط ​​معدل الذكاء للأطفال65 و معظمهم نسبته من بسيط إلى شديد ولكن لا يوجد فرق ذو دلالة إحصائية بينه وكذلك بين نتائج رسم المخ وشدة أعراض التوحد باستخدام اختبار كارز.

**الخلاصة:** يعتبر اضطراب طيف التوحد هو اضطراب فى نمو الجهاز العصبي للطفل وطرق الآتصال بينه و لا يوجد اتفاق على ملامح العلاقه بين اضطراب التوحد ونتائج رسم المخ على الرغم من أن بعض الدراسات يتفقون على ارتفاع معدل انتشار التشوهات الصرعيه في الأطفال الذين يعانون من التوحد.

**الكلمات المفتاحية:** اضطراب طيف التوحد, رسم المخ الكهربائى.